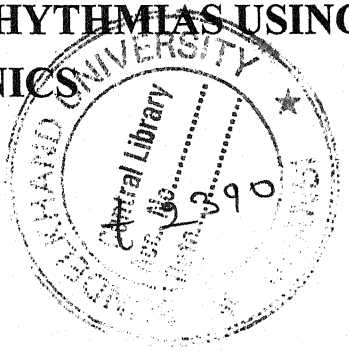
SIMULATION OF NORMAL HUMAN ECG (ELECTROCARDIOGRAPH) AND ARRHYTHMIAS USING ADVANCED ELECTRONICS



THESIS

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DOCTOR OF PHILOSOPHY

IN

BIOMEDICAL ENGINEERING

By

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2007

CERTIFICATE

This is to certify that the thesis entitled ***"Simulation of Normal Human ECG (Electrocardiograph) and Arrhythmias using Advanced Electronics"*** is a piece of research work done by Er. Shahanaz Ayub under our supervision in the department of Biomedical Engineering, Institute of Engineering & Technology, Bundelkhand University, Jhansi for the award of the degree of ***Doctor of Philosophy*** in Biomedical Engineering. She has put in an attendance of more than two hundred days as required by the Bundelkhand University ordinance during the research period.

To the best of my knowledge and belief the thesis :

(i) embodies the work of the candidate herself.

(ii) has duly been completed.

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Declaration

I hereby declare that the thesis entitled **"Simulation of Normal Human ECG (Electrocardiograph) and Arrhythmias using Advanced Electronics"** being submitted for the degree of **"Doctor of Philosophy"** to the Bundelkhand University, Jhansi, is an innovative piece of work which has been carried out by me at Department of Biomedical Engineering, Institute of Engineering & Technology, Bundelkhand University, Jhansi (U.P.).

To the best of my knowledge and belief it has not been submitted in part or full for any other degree.

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(Shahanaz Ayub)

ABSTRACT

Cardiac Monitoring is now an essential and important medical field. It includes diagnosis and continuous monitoring of patients suffering from cardiac troubles. As the monitoring facility increases, it is also important to check whether the machines are functioning properly, so that they produce correct patient data. For this purpose simulators were introduced, which gave reliable output which represented normal or abnormal patient data. This data was given to the machine and checked whether the machine produced the expected waveforms. With further development simulators which could be used as tool of comparison for intelligent monitor to diagnose the patient data and give directly the disease or arrhythmia the patient is suffering from could also be developed.

The hospital coronory care unit was developed to provide the critical cardiac patient with the specialised attention of highly skilled medical professionals aided by cardiac surveillance equipment. Detection and treatment of arrhythmias has become one of the CCU's (Cardiac Care Unit) major functions.

A close correlation exists between innovative developments and enhanced prognosis and patient longevity. Today the CCU patient may require constant and exact monitoring that is provided by specially programmed, sophisticated arrhythmia / ECG (Electrocardiograph) monitors. These instruments can detect with a high degree of accuracy potentially life threatening arrhythmias thus alerting the hospital staff to the patient's need for immediate treatment.

The diagnostic ECG is usually recorded in the form of orthogonal leads, such as Frank leads or 12-lead recordings. The orthogonal lead system provides a readily interpretable picture of a cardiac vector, but the 12-lead system is now more popular as considerable clinical expertise has been acquired in interpreting the waveforms that appear in 12-lead ECGs. The instrumentation must produce high fidelity recordings followed by high-resolution digitization. Full 12-lead analysis makes certain tasks, such as reliable QRS detection, more reliable. At the same time extensive rules and algorithms arte needed to classify complex arrhythmias. Interpretation based on simultaneous recordings from several leads is helpful in diagnosing conditions such as hypertrophy or

myocardial infarction. The diagnostic ECG systems are useful in cardiologists offices, as portable carts in bedside recording, as patient monitors in cardiac intensive care, and so on. Modern commercial systems are based on multiple microprocessors that do sophisticated realtime ECG data acquisition and analysis. A number of monitoring systems may be connected together as in intensive care or via telephone or communication networks. They share sophisticated interpretation algorithms and large database. Diagnosis by the automated ECG interpretation system is usually not perfect and must be validated by human observers, Still ECG interpretation by computers reduce costs and makes ECG interpretation services widely available.

Arrhythmia / ECG simulator is a device for quick and accurate testing of arrhythmia / ECG monitors. This unit allows the user to ascertain that the arrhythmia / ECG monitor is recognising normal ECG and typical arrhythmia and properly responding when such signals occur. The instrument is therefore, an essential tool for CCU personnel.

Today the available simulators are for normal ECG or separately arrhythmia simulator detecting only LEAD II signal and not the abnormalities of all points.

Hence the need is for ECG / Arrhythmia simulator which simulate the abnormalities of all point waveforms e.g. LA, RA, LL, V1, V2, V3, V4, V5 and V6. Present Arrhythmia monitors does give the interpretations of the arrhythmias but the arrhythmia patterns are stored in the software and not accessible. Further its use is restricted to the Arrhythmia monitors only. Hence one must know the surety of the interpretations given by these monitors before preparing the patient's report.

Till date no such simulator was designed to provide a means to confirm these interpretations.

The research work consists of designing of the ECG / Arrhythmia simulator.

The thesis reviews about the structure (anatomy) of the heart which is the source of ECG, Conduction System of the heart i.e. how the impulse is generated and conducted to various parts of the heart and body and how the electrocardiogram (ECG) is generated. This also describes about the details of the ECG waves, their amplitude, timings and abnormalities. It also gives the information about the arrhythmias, What are these, their

classification, description, features, symptoms and causes about major life threatening arrhythmias.

Based on mathematical modelling and leads theory, using the equations for potentials of the 12 lead ECGs, a wide database is generated for Normal human ECG and seventeen arrhythmias (abnormal ECG). This type of simulation is implemented on EXCEL and the Lead II graphs are plotted.

The simulation of ECG / Arrhythmias is done using various techniques. Simulation using Microcontroller 8752 gives the development work and portable unit to carry at any Cardiac Care Unit for monitoring proper functioning of the ECG / Arrhythmia monitors. It gives the details of designing circuits, its software, its implementation and results.

The Window based software ECGSIM is used for simulating ventricles response i.e. QRS potentials. The Window tools are explained in detail as well as features of ECGSIM, the simulation of Heart pane, Thorax pane, Membrane pane, ECG pane is also described.

The results obtained give satisfactory performance reports of this simulation process. Also the simulation results are compared with the actual patients 12 lead signals. The research work achieves the objectives targeted and is useful in the field of cardiology and biomedical engineering.

CONTENTS

			Page No.
ACKNOWLEDGEMENTS			
ABSTRACT			i-iii
TABLE OF CONTENTS			iv-vii
LIST OF ABBREVIATIONS			viii
LIST OF SYMBOLS			ix
LIST OF FIGURES			x-xiii
LIST OF TABLES			xiv
Chapter-I	**INTRODUCTION**		
	1.1	GENERAL	1
	1.2	SOME IMPORTANT APPLICATIONS OF ECG	3
	1.3	BASE OF DIAGNOSTIC ECG INTERPRETATION	3
	1.4	STEPS INVOLVED IN DEVELOPING ALGORITHMS BY COMPUTER AIDED ANALYSIS	5
	1.5	MOTIVATION TO THE RESEARCH WORK	7
	1.6	OBJECTIVE OF THE WORK	8
	1.7	DISSECTION OF THESIS	9
	1.8	CONCLUSION	10
Chapter-II	**LITERATURE REVIEW**		
	2.1	INTRODUCTION	11
	2.2	THE HEART	11
		2.2.1 LOCATION AND SIZE OF THE HEART	12
		2.2.2 BASIC ANATOMY OF THE HEART	12
	2.3	CONDUCTION SYSTEM	18
		2.3.1 AUTORHYTHMIC CELLS	18
		2.3.2 TIMING OF ATRIAL AND VENTRICULAR EXCITATION	19
		2.3.3 PHYSIOLOGY OF CARDIAC MUSCLE CONTRACTION	20

	2.3.4	CARDIAC ACTION POTENTIAL	21
	2.3.5	PACEMAKER TISSUES	25
	2.3.6	(SINGLE-FIBER) ELECTRO-PHYSIOLOGICAL PRINCIPLES	30
	2.3.7	CARDIAC SOURCES	35
	2.3.8	EQUIVALENT SINGLE DIPOLE	38
2.4.		GENERATION OF ECG AND ECG WAVES	41
	2.4.1	THE NORMAL ECG WAVE	43
	2.4.2	ECG RECORDING CONVENTIONS	44
	2.4.3	WAVES ASSOCIATED WITH ECG	44
2.5		ARRHYTHMIA	48
	2.5.1	MECHANISMS THAT CAUSE ABNORMAL IMPULSES	48
	2.5.2.	CAUSES OF ARRHYTHMIAS	49
	2.5.3	CLASSIFICATION OF ARRHYTHMIAS	50
	2.5.4	FUNDAMENTAL DESCRIPTIVE PROPERTIES OF CARDIAC RHYTHMS	51
	2.5.5	SOME IMPORTANT ARRHYTHMIAS	53
	2.5.6.	SUMMARY OF COMMONLY OCCURRING LIFE THREATENING ARRHYTHMIAS	65
2.6		CONCLUSION	68
CHAPTER - III		**SIMULATION USING MATHEMATICAL MODELLING OF THE HEART AND LEADS THEORY**	
3.1.		INTRODUCTION	69
3.2		ECG LEADS	69
	3.2.1	LIMB LEADS	70
	3.2.2	AUGMENTED LIMB LEADS	73
	3.2.3	PRECORDIAL LEADS	74
	3.2.4	GROUND LEAD	76
	3.2.5	CLINICAL LEAD GROUP	76
3.3		EXPERIMENTAL WORK	78
3.4		CONCLUSION	130

CHAPTER –IV ECG/ARRHYTHMIA SIMULATOR USING MICROCONTROLLER

4.1 INTRODUCTION 131

4.2 DESIGN 132

4.2.1 DESIGNING OF POWER SUPPLY 133

4.2.2 DESIGNING OF DIGITAL SECTION 137

4.2.3 DESIGNING OF ANALOG SECTION 141

4.2.4. KEYBOARD AND DISPLAY SECTION 144

4.3 SOFTWARE 147

4.3.1 ALGORITHM AND FLOWCHARTS 147

4.3.2 HEX CODES USED IN SOFTWARE 152

4.3.3 LOOK UP TABLES 152

4.4 PCB LAYOUT AND FABRICATION 162

4.4.1 MICROCONTROLLER AND ANALOG SECTION 162

4.4.2 KEYBOARD AND DISPLAY SECTION 163

4.5 TESTING OF PCBs 163

4.6 RESULTS & CONCLUSION 170

4.6.1 HOLTER CARDIOGRAPHY REPORT 171

CHAPTER – V ECGSIM PROGRAM FOR QRS SIMULATION

5.1 INTRODUCTION 181

5.2 ECGSIM CONTENTS 181

5.3 ECGSIM ALGORITHMS 182

5.4 BASIC USAGE OF ECGSIM WINDOW 183

5.4.1 FILE 184

5.4.2 FILE FORMATS 186

5.5 HEART PANE 189

5.5.1 SURFACE FUNCTION 190

5.5.2 DISPLAY OPTIONS 190

5.5.3 SCALE 191

5.5.4 MOUSE ACTIONS IN THE HEART PANE 192

5.6 THORAX PANE 192

5.6.1 SURFACE FUNCTION 193

		5.6.2 DISPLAY OPTIONS	193
		5.6.3 SCALE	194
		5.6.4 MOUSE ACTIONS IN THE THORAX PANE	195
	5.7	MEMBRANE PANE	195
	5.8	ECGs PANE	200
	5.9	MOVIE	203
	5.10	HELP	203
	5.11	ALTERNATIVE GEOMETRY	204
	5.12	CONCLUSION	206
CHAPTER-VI	**RESULTS AND CONCLUSION**		
	6.1	INTRODUCTION	208
	6.2	RESULTS	208
	6.3	COMPARISON OF THE SIMULATED REPORTS WITH THE ACTUAL PATIENTS ECG SIGNALS	209
	6.4	CONCLUSION	219
CHAPTER-VII	**FURTHER SCOPE OF WORK**		221
	REFERENCES		224
	LIST OF PUBLICATIONS		233
	APPENDIX-I		
	APPENDIX-II		

LIST OF ABBREVIATIONS

AC	-	Alternating current.
AH	-	Ampere Hour.
AV	-	Atrio Ventricular .
aV_F	-	The potential at the left leg using both arms to form the indifferent electrode.
aV_L	-	The potential at the left arm using the right arm and left leg to form the indifferent electrode.
AVN	-	Atrio Ventricular Node.
aV_R	-	The potential at the right arm using the left arm and left leg to form the indifferent electrode.
CCU	-	Cardiac Care Unit.
CT	-	Central Terminal
DAC	-	Digital to Analog Converter.
DC	-	Direct Current.
DEMUX	-	Demultiplexer.
ECG	-	Electrocardiogram.
IC	-	Integrated circuit.
IVC	-	Inferior Vena Cava.
LA	-	Left Arm.
LAD	-	Left Anterior Descending.
LED	-	Light Emitting Diode.
LL	-	Left Leg.
OPAMP	-	Operational amplifier.
RA	-	Right Arm.
SA	-	Sino Atrial.
SAN	-	Sino Atrial Node.
SVC	-	Superior Vena Cava.
SVPB	-	Supraventricular Premature Beats.
SVT	-	Paroxysmal supraventricular Tachycardia.
V_F	-	Non augmented unipolar left leg voltage.
V_L	-	Non augmented unipolar left arm voltage.
VLSI	-	Very Large Scale Integrated Circuit.
V_R	-	Non augmented unipolar right arm voltage .
µC	-	Microcontroller.

LIST OF SYMBOLS

Vm	=	Transmembrane potential in milivolts.
[K]e	=	Extracellular potassium concentration.
[k]i	=	Intracellular potassium concentration.
[Na]e	=	Intracellular sodium concentration.
[Na]i	=	Intracellular sodium concentration
Vm(t)	=	Temporal action potential
Vm(z-θt)	=	Spacio temporal behaviour
θ	=	Velocity of propagation along z axis
ϕ_I	=	Intracellular potential
i_m	=	Transmembrane current per unit length
E	=	Extracellular electrical field
J	=	Extracellular current density
σ_e	=	Extracellular conductivity
W	=	Source layer density of each layer
D	=	Separation of layers
τ	=	Dipole density strength
Wds	=	Point source
R^+	=	Source field distance from elements on the positive layer
R^-	=	Source field distance from elements on the negative layer
Ω	=	Solid angle subtended at the field point by the associated double layer
V	=	Volume
S	=	Surface

LIST OF FIGURES

Sr. No.		Figures	Page No
1	2.1	Structure of the heart.	13
2	2.2	Impulse (Action Potential) in a ventricular Contractile Fiber.	20
3	2.3	Cardiac Action Potential Phases	23
4	2.4	Conduction System of the Heart	28
5	2.5	Stimulating Current Flow	33
6	2.6	Linear Core conductor model of an excitable fiber	34
7	2.7	Evaluation of cardiac sources	36
8	2.8	Normal ECG Waveform (Lead II)	42
9	2.9	Graphic representation of the use of the 'ladder diagram in depicting arrhythmias'	53
10	2.10	Arrhythmia Waveforms (Lead II)	67
11	3.1	Graphic showing the relationship between positive electrodes	69
12	3.2	Limb Leads	70-71
13	3.3	Proper Placement of the Limb Leads	72
14	3. 4	Proper placement of the precordial leads.	74
15	3. 5	The Contiguous leads	76
16	3. 6	Diagram showing how the polarity of the QRS complex in leads I, II, and III can be used to estimate the heart's electrical axis in the frontal planec	77
17	3.7	Types of the connections with typical ECG waveform (a) bipolar timb leads (b) unipolar leads	79
18	3.8	Types of the connections with typical ECG waveform (c) position of the chest lead in unipolar precordial lead recording (d) C leads	80
19	3. 9	Normal ECG lead II wave	81
20	3.10	Bradycardia lead II wave	82
21	3.11	Tachycardia lead II wave	89

22	3.12	Missed beat lead II signal	92
23	3.13	Atrial fibrillation lead II signal	96
24	3.14	Ventricular fibrillation lead II signal	100
25	3.15	Bigeminy lead II signal	100
26	3.16	Couplet lead II signal	105
27	3.17	Run lead II signal	109
28	3.18	Multifocal run lead II signal	113
29	3.19	R on T wave lead II signal	117
30	3.20	Ventricular tachycardia lead II signal	119
31	3.21	Heart block lead II signal	123
32	3.22	VPB 1 lead II signal	123
33	3.23	VPB 2 lead II signal	126
34	4.1	Block Diagram of ECG/Arrhythmia Simulator using Microcontroller.	131
35	4.2	Power Supply Circuit for ECG/Arrhythmia Simulator using Microcontroller.	134
36	4.3	Circuit Diagram of ECG/Arrhythmia Simulator using microcontroller – Digital Section	138
37	4.4	Circuit Diagram of ECG/Arrhythmia Simulator using Microcontroller – Analog section	139
38	4.5	Flowchart – Initialisation Program of ECG/Arrhythmia Simulator using Microcontroller	149
39	4.6	Generalised Flowchart To detect Arrhythmia using microcontroller	150
40	4.7	Flowchart of arrhythmia (Tachycardia) using microcontroller	151
41	4.8	PCB layout solder side of ECG/Arrhythmia simulator using microcontroller	164
42	4.9	PCB layout component side of ECG/Arrhythmia simulator using microcontroller	165
43	4.10	PCB layout Silk Screen side of ECG/Arrhythmia simulator using microcontroller	166
44	4.11	PCB layout Solder side of keyboard and display section of	167

		ECG/ Arrhythmia simulator using microcontroller	
45	4.12	PCB layout - Component side of keyboard and display section of ECG/ Arrhythmia simulator using microcontroller.	168
46	4.13	PCB layout – Silk Screen of keyboard and display section of ECG/ Arrhythmia simulator using microcontroller.	169
47	4.14	Arrhythmia simulation report 1 obtained at Nair Hospital, Mumbai on Holter cardiography	172
48	4.15	Arrhythmia simulation report 2 obtained at Nair Hospital, Mumbai on Holter cardiography	173
49	4.16	Arrhythmia simulation report 3 obtained at Nair Hospital, Mumbai on Holter cardiography	174
50	4.17	Arrhythmia simulation report 4 obtained at Nair Hospital, Mumbai on Holter cardiography	175
51	4.18	Arrhythmia simulation report 5 obtained at from cardiomin monitor	176
52	4.19	Arrhythmia simulation report 6 obtained at from cardiac monitor.	177
53	4.20	Arrhythmia simulation report 7 obtained at from cardiac monitor.	178
54	4.21	ECG / Arrhythmia simulation report 8 obtained on BPL 108T ECG Machine (Lead V1 Waveforms of normal ECG and 17 Arrhythmias)	179
55	4.22	ECG /Arrhythmia simulator using microcontroller.	180
56	5.1	ECGSIM Window	182
57	5.2	The Heart Pane	189
58	5.3	The Membrane Pane	196
59	5.4	The ECG Pane	201
60	6.1	12 Lead ECG signal of an 84 year old lady with hypertension	209
61	6.2	12 Lead ECG signal of a lady with Romano-Ward Syndrome.	210
62	6.3	12 Lead ECG signal of a normal adult	211

63	6.4	12 Lead ECG signal of a 47 year old man with a long history of palpitations and lately, blackouts.	212
64	6.5	12 Lead ECG signal of a 45 year old lady with palpitations and history of chronic renal failure	213
65	6.6	12 Lead ECG signal of a 72 year old man with a permanent pacemaker	214
66	6.7	12 Lead ECG signal of A 60 year old man with 2 hours of "crushing" chest pain suddenly collapses.	215
67	6.8	12 Lead ECG signal of a 34 year old lady with asthma	216
68	6.9	12 Lead ECG signal of a 55 year old man with 4 hours of "crushing" chest pain	216
69	6.10	12 Lead ECG signal of a 59 year old lady with chronic bronchitis	217
70	6.11	12 Lead ECG signal of a 76 year old man with breathlessness.	218
71	6.12	12 Lead ECG signal of a 58 year old man on haemodialysis presents with profound weakness after a weekend fishing trip.	218

LIST OF TABLES

Sr. No.	Tables		Page No.
1	Table 1.1	Various interpretations of ECGs based on computer aided analysis.	6
2	Table 2.1	Intracellular and extracellular ion concentrations	22
3	Table 2.2	Conduction speed in Cardiac Tissue.	28
4	Table 3.1	Normal ECG lead II Data	82-85
5	Table 3.2	Bradycardia lead II data	86-88
6	Table 3.3	Tachycardia lead II data	90-92
7	Table 3.4	Missed beat data	93-96
8	Table 3.5	Atrial fibrillation data	97
9	Table 3.6	Ventricular Fibrillation data	98-99
10	Table 3. 7	Bigeminy data	101
11	Table 3.8	Couplet data	102-105
12	Table 3.9	Run data	105-109
13	Table 3.10	Multifocal Run data	110-113
14	Table 3.11	R on T Wave data	114-117
15	Table 3.12	Ventricular Tachycardia data	118-119
16	Table 3.13	Heart Block Data	120-122
17	Table 3.14	VPB 1 (Ventricular Premature Beat 1) data	124-126
18	Table 3.15	VPB2 (Ventricular Premature Beat2) data	127-130
19	Table 4.1	LED Configuration	146
20	Table 4.2	The data for normal ECG signal	152-157
21	Table 4.3	Data for vpb1	157-159
22	Table 4.4	Data for atrial firbrillation	159
23	Table 4.5	Data for ventricular fibrillation	159-162

CHAPTER - I

INTRODUCTION

1.1 GENERAL

A medical topic that attracted the early interest of engineers is that of electrocardiography. It is one of the most useful noninvasive medical diagnostic tests and is in very wide use (an estimated 200 million ECGs taken each year). Its technological challenge is to improve performance by increasing the number of simultaneously recorded signals, raising the signal to noise ratio, and accomplishing these goals, possibly, with the subject undergoing physical activity. The increase in data acquisition rate also provides new challenges in the design of recording systems including the application methods of data compression. In addition, the analysis of these voluminous data has been approached through the application of a variety of automated pattern recognition techniques.

On a more fundamental level, Biomedical engineering introduced a quantitative description of the electrical sources (generators) in the heart that are responsible for the ECG, ECG i.e. electrocardiogram is the electrical activity of the heart and when it is abnormal called arrhythmia. Through the application of the principles of electromagnetic theory, the fields generated by these sources at the surface of the torso (considered as an in homogeneous volume conductor) have been quantitatively evaluated, and in this way the effects of geometry, electrical conductivity, electrophysiology, and most importantly, pathology are being studied. The research work presented in this thesis provides an increased ability to clinically interpret electrocardiographic signals beyond the essentially statistical approach prevalent today. In particular, the theoretical approaches to the inverse problem in mathematical physics are being adapted and tested in the electrocardiographic context.

The research on electrocardiography is organized to present the current state of knowledge from a logical (rather than historical) approach. It begins with an introduction to the principles underlying the electrical activity of a single cell to include a description of the associated electrical sources in engineering (i.e. quantitative) terms. A typical,

observed electrical behavior of the heart is then presented. A generalization of the single cell electrophysiology is then made from which a quantitative description of cardiac sources of the whole heart is formulated. The fields arising in the volume conductor due to these sources is also described. Based on this model the nature of the electrocardiographic field that is sampled at the torso surface, using a placement electrodes that has arisen through clinical experience, is described. With this as the basic structure, the remaining questions are approached in their strictly engineering contest. These deal with simulation of ECG (Electrocardiogram) Normal and abnormal (Arrhythmia) with various techniques using mathematical model, microcontroller programming, and a windows based software ECGSIM and the recorded electrocardiographic waveforms.

The hospital coronary unit was developed to provide the critical cardiac patient with the specialized attention of highly skilled medical professionals, aided by cardiac surveillance equipment. Detection and treatment of arrhythmias has become one of the CCU's major functions.

A close correlation exists between innovative developments and enhanced prognosis and patient longevity. Today the CCU patient may require constant and exact monitoring that is provided by specially programmed, sophisticated Arrhythmia / ECG monitors. These instruments can detect with a high degree of accuracy potentially life threatening arrhythmias, thus alerting the hospital staff to the patients need for immediate treatment.

Cardiac arrhythmias are often first detected by simple but nonspecific means : auscultation of the heartbeat with a stethoscope, or feeling for peripheral pulses. These cannot diagnose specific arrhythmias, but can give a general indication of the heart rate and whether it is regular or irregular. Not all the electrical impulses of the heart produce audible or palpable beats; in many cardiac arrhythmias, the premature or abnormal beats do not produce an effective pumping action and are experienced as "skipped" beats.

The simplest specific test for assessment of heart rhythm is the electrocardiogram [1] (abbreviated ECG or EKG). A Holter monitor is an ECG recorded over a 24-hour period, to detect arrhythmias that may happen briefly and unpredictably throughout the day.

The ECG has become so familiar to the general population that it is part of the logo of many medical organisations, representing the technical side of medicine vs. the Rod of Asclepius or caduceus, which are more traditional. Being an electrical representation, it signifies vitality and urgency.

An isoelectric ECG (no cardiac electrical activity or flatline) is often used as a symbol of death or at least extreme medical peril. This is technically known as asystole, a form of cardiac arrest with a particularly bad prognosis.

1.2 SOME IMPORTANT APPLICATIONS OF ECG

- Determine whether the heart is performing normally of suffering from abnormalities (eg. extra or skipped heartbeats- cardiac arrhythmia).
- May indicate acute or previous damage to heart muscle (heart attack) or ischaemia of heart muscle (angina)
- Can be used for detecting potassium, calcium, magnesium and other electrolyte disturbances.
- Allows the detection of conduction abnormalities (heart blocks and bundle branch blocks)
- As a screening tool for ischaemic heart disease during an exercise tolerance test.
- Can proved information on the physical condition of the heart (eg : left ventricular hypertrophy, mitral stenosis)
- Can suggest non-cardiac disease (e.g. pulmonary embolism, hypothermia)

1.3 BASE OF DIAGNOSTIC ECG INTERPRETATION

Diagnostic ECG interpretation [2] requires high-quality signals (that is, noise-free signals with proper gain and band-width) recorded from several channels. Therefore, the algorithms shall take into consideration the redundancy available in many leads. For example, the QRS complex may be isoelectric in one lead, but may be quite large in an orthogonal lead. Artifact is often not present in all the leads at the same time. This is because the artifact often arises from electrode failure or movement at some specific

electrode site. Thus, artifact can be rejected by selectively processing the leads. The ECG interpretation algorithms [3], to a large extent, rely first on accurate QRS detection. Some arrhythmias such as bradycardia can be classified simply from beat-to-beat intervals. The QRS detector also helps localize the exact incidence in time of the Q, R, S, P, and T waves for later morphologic analysis. Ectopic beats, such as paced or fusion beats, may be classified on the basis of beat morphology. This also makes the task of detecting some arrhythmias, such as atrial fibrillation (characterized by high rate of atrial depolarization) or atrioventricular dissociation (discontinuous conduction between atria and ventricles), quite difficult. P-wave detection is crucial in identification of atrial disorders. Analysis of ST segment and the T wave is particularly useful in the detection of myocardial ischemia and infarction. Classification of more complex arrhythmias depends on a combination of rhythm and morphology analysis.

Computer-aided analysis [3] of abnormal ECG patterns first requires quantification of specific features such as the amplitude, duration, polarity, and so on. Beat morphologies manifest themselves differently in different leads. Therefore, such an analysis must be simultaneously carried out in several leads. ECG interpretation rules are derived by careful selection of criteria that must be violated for identification of specific disorders. The rules themselves must be finally transcribed in the form of flow charts, decision tables, and programs.

The software development process begins by devising either flowcharts or decision tables. Flowcharts are commonly employed in many software application where the computer goes through a series of logical actions. The program flow may depend on the result of specific tests. Such decision points are illustrated by diamonds in the flow-chart. The principal criteria for arrhythmia detection are R-R interval and heart rate. The flowchart distinguishes a few arrhythmia groups on the basis of these criteria. More relined discussion would require additional criteria and tests.

Diagnostic ECG interpretation deals with a large number of disorders, and requires more information than just the rate for decision making. For example, the pattern of the ECG waveform itself must be analyzed in more detail. For example, analyzes parts of the QRS complex. On the basis of slope the table determines if the QRS complex has an RS or QR configuration.. Once again a decision table lists all the criteria associated with a specific group of disorders. When all the criteria are specified, a specific disorder such as acute inferior infarction is recognized. It becomes clear that a large number of such tables are required to diagnose important disorders that a detailed diagnostic ECG program should be able to interpret.

1.4 STEPS INVOLVED IN DEVELOPING ALGORITHMS BY COMPUTER AIDED ANALYSIS

1. ECG filtering and artifact rejection
2. QRS detection and rate calculations
3. Identification of P and T waves and ST segments
4. Ectopic beat identification (PVC, paced, fusion. etc.)
5. Arrhythmia classification based on rate (bradycardia, tachycardia, etc.)
6. Classification of disorders based on morphology (hypertrophy, infarct. etc.)

The performance of the computer programs [3] for diagnostic ECG needs to be verified. This is usually accomplished by concurrent review of patient ECG [4] by a trained cardiologist. It is commonly believed, though, that there are widespread inconsistencies and disagreements among cardiologists. Consequently, some standardization would be desirable. A database annotated by a panel of experts, so-called gold standard; would find a wider acceptance. Such a gold standard could be used for the evaluation of diagnostic ECG computer programs. Currently, no such database is available for 12-lead ECGs. An annoted data base for 12 lead ECGs comprising recordings from Holter tapes and cardiac intensive care patients has now been developed by the American Heart Association [5]. Considerable efforts have gone into validation of many computer programs available from commercial institutions. There is a wide variability among these programs [6]. Table 1.1 source the various interpretations of ECGs based on computer aided analysis.

Table 1.1 : Various interpretations of ECGs based on computer aided analysis.

1.	Normal sinus rhythm	2.	Sinus bradycardia
3.	Sinus tachycardia	4.	Junctional rhythm
5.	Wandering atrial pacemaker	6.	Sinus arrhythmia
7.	Atrial fibrillation	8.	Attrial flutter
9.	Premature atrial contraction	10.	Premature ventricular contraction
11.	AV dissociation	12.	First-degree AV block
13.	Second-degree AV block	14.	Third-degree AV block
15.	Pacemaker	16.	Prolonged QT duration
17.	Right-axis deviation	18.	Left-axis deviation
19.	RBBB complete or incomplete	20.	LBBB
21.	Ventricular conduction delay	22.	Left anterior hemiblock
23.	Left posterior hemiblock	24.	WPW-type A
25.	WPW-type B	26.	Right atrial enlargement
27.	Left atrial enlargement	28.	Right ventricular hypertrophy
29.	Prominent anterior forces	30.	Prominent left ventricular voltage
31.	Left ventricular hypertrophy	32.	Left ventricular hypertrophy with strain
33.	Poor R-wave progression in precordial leads	34.	Anterior or anteroseptal myocardinal infraction (MI)
35.	Anteriolateral MI	36.	Inferior or inferior apical MI
37.	Posterior MI	38.	Possible lateral MI
39.	Possible inferior MI	40.	T-wave abnormality
41.	ST segment elevation	42.	ST segment elevation
43.	Minimal repolarisation variant	44.	Nonspecific S-T and T changes

1.5 MOTIVATION TO THE RESEARCH WORK

The diagnostic ECG is usually recorded in the form of orthogonal leads, such as Frank leads or 12-lead recordings. The orthogonal lead system provides a readily interpretable picture of a cardiac vector, but the 12-lead system is now more popular as considerable clinical expertise has been acquired in interpreting the waveforms that appear in 12-lead ECGs. The instrumentation must produce high fidelity recordings [7] followed by high-resolution digitization. Full 12-lead analysis makes certain tasks, such as reliable QRS detection, more reliable. At the same time extensive rules and algorithms are needed to classify complex arrhythmias. Interpretation based on simultaneous recordings from several leads is helpful in diagnosing conditions such as hypertrophy or myocardial infarction. The diagnostic ECG systems are useful in cardiologists offices, as portable carts in bedside recording, as patient monitors in cardiac intensive care, and so on. Modern commercial systems are based on multiple microprocessors that do sophisticated realtime ECG data acquisition and analysis. A number of monitoring systems may be connected together as in intensive care or via telephone or communication networks. They share sophisticated interpretation algorithms [2] and large database. Diagnosis by the automated ECG interpretation system is usually not perfect and must be validated by human observers, Still ECG interpretation by computers reduce costs and makes ECG interpretation services widely available.

Arrhythmia / ECG simulator is a device for quick and accurate testing of arrhythmia/ECG monitors. This unit will allow the user to ascertain that the arrhythmia/ ECG monitor is recognizing normal ECG and typical arrhythmia and properly responding when such signals occure. The instrument is therefore, an essential tool for CCU personnel.

Today the available simulators [8] are for normal ECG or separately arrhythmia simulator detecting only lead II signal and not the abnormalities of all points.

Hence the need is for ECG and Arrhythmia simulator which simulate the abnormalities of all point waveforms i.e. LA, RA,LL, V1, V2, V3, V4, V5, V6.

Present Holter cardiography [9] does give the interpretation of the arrhythmias but the arrhythmia patterns are stored in the software and not accessible. Further its use is restricted to the holter equipment only, hence one must know the surety of the

interpretation given by Holter cardiography before preparing the report of the patient. ECG / Arrhythmia simulator provides a means to confirm these interpretations.

The seventeen different life threatening arrhythmias selected for simulation to obtain Lead I, Lead II, Lead III, aV_R, aV_L, aV_F, V1, V2, V3, V4, V5, V6 are Bradycardia, Tachycardia, Asystole, Fusion beat, Missed beat, Atrial fibrillation, Ventricular fibrillation, Bigeminy, Multifocal Ventricular Extrasystoles, R on T wave, Ventricular Tachycardia, Heartblock, VPB1 and VPB2.

1.6 OBJECTIVE OF THE WORK

Diagnostic ECG interpretation requires high-quality signals (that is, noise-free signals with proper gain and band-width) recorded from several channels. The present algorithms take into consideration the redundancy available in many leads. For example, the QRS complex may be isoelectric in one lead, but may be quite large in an orthogonal lead. Artifact is often not present in all the leads at the same time. This is because the artifact often arises from electrode failure or movement at some specific electrode site. Thus, artifact can be rejected by selectively processing the leads. The ECG interpretation algorithms, to a large extent, rely first on accurate QRS detection. Some arrhythmias such as bradycardia can be classified simply from beat-to-beat intervals. The QRS detector also helps localize the exact incidence in time of the Q, R, S, P, and T waves for later morphologic analysis. Ectopic beats, such as paced or fusion beats, may be classified on the basis of beat morphology. This also makes the task of detecting some arrhythmias, such as atrial fibrillation (characterized by high rate of atrial depolarization) or atrioventricular dissociation (discontinuous conduction between atria and ventricles), quite difficult. P-wave detection is crucial in identification of atrial disorders. Analysis of ST segment and the T wave is particularly useful in the detection of myocardial ischemia and infarction. Classification of more complex arrhythmias depends on a combination of rhythm and morphology analysis. The following objectives have been laid down for this research work.

The objective of the work is :

1. To simulate noise free high quality signals at RA, LA, LL, V1, V2, V3, V4, V5, V6 for normal ECG and seventeen arrhythmias as Bradycardia, Tachycardia,

Asystole, Fusion beat, Missed beat, Atrial fibrillation, Ventricular fibrillation, Bigeminy, Multifocal Ventricular Extrasystoles, R on T wave, Ventricular Tachycardia, Heartblock, VPB1 and VPB2.

2. To develop an easily operated window based simulation for teaching purpose and research purpose.

3. To develop the portable unit of ECG/Arrhythmia simulator battery operated so as to carry at any CCU to check the ECG/Arrhythmia monitor performance.

4. To generate a database of Normal ECG and seventeen arrhythmias.

1.7 DISSECTION OF THESIS

The present thesis embodies the investigations on the specific aspects as listed in the previous section. The thesis is organized into seven chapters and contents of each chapter are presented in brief as follows :

Chapter II, reviews and also describes about the structure (anatomy) of the heart which is the source of ECG, Conduction System of the heart i.e. how the impulse is generated and conducted to various parts of the heart and body and how the electrocardiogram (ECG) is generated. This also describes about the details of the ECG waves, their amplitude, timings and abnormalities. This chapter also gives the information about the arrhythmias, What are these?, their classification, description, features, symptoms and causes about major life threatening arrhythmias which is of clinical significance to the cardiologist.

In chapter III, based on mathematical modeling and using the equations for potentials of the 12 lead ECGs, a wide database is generated for Normal human ECG and seventeen arrhythmias (abnormal ECG). This type of simulation is implemented on EXCEL and the Lead II graphs are plotted. This chapter also describes about ECG leads and leads theory.

Chapter IV describes the simulation process of ECG / Arrhythmias simulation using Microcontroller 8752. This chapter describes about the experimental work to develop a portable unit to carry at any Cardiac Care Unit for monitoring proper functioning of the ECG / Arrhythmia monitors. It gives the details of designing circuits, its software, its implementation, testing and results.

Chapter V describes the Windows based software ECGSIM for simulating ventricles response i.e. QRS potentials. The Window tools are explained in detail as well as features of ECGSIM, the simulation of Heart pane, Thorax pane, Membrane pane, ECG pane is also described.

Chapter VI describes the results and gives the satisfactory performance reports of this simulation process. Also it gives the comparison of this simulation waveforms (simulated patients) with the actual patients 12 lead ECG signals. The patients are belonging to different age groups, females, males and having different health problems. This chapter also concludes the research studies and also brings out significant and important contributions of this Ph.D. thesis.

Finally in chapter VII a short but comprehensive accounts of further extensions of this research is presented.

1.8 CONCLUSION

This chapter provides an overview of the research work carried out and embedded in this dissertation. The wide usage of ECG and the base of ECG interpretation is discussed. The objective and motivation of the work is defined. This has resulted in defining scope of the research work.

CHAPTER - II

LITERATURE REVIEW

2.1 INTRODUCTION

The heart [10] is the centre of the cardiovascular system. Where as the term cardio refers to the heart, the term vascular refers to blood vessels (or an abundant blood supply). The heart propels blood through thousands of miles of blood vessels, and it is magnificently designed for this task. Although we ignore its activity most of the time, the heart's capacity work is remarkable. Even at rest, the heart pumps 30 times its own weight each minute, about 5 liters to the lungs and the same volume to the rest of the body. At this rate the heart would pump more than 7000 liters of blood in a day and 5 million liters in a year. Since we don't spend all our time "resting" and since our heart pumps more vigorously when we are active, the actual flow is much larger.

The cardiovascular system provides the "pump" for circulating constantly refreshed blood through an estimated 100,000 Km of blood vessels. As blood flows through body tissues, nutrients and oxygen move from the blood into the interstitial fluid and then into cells. At the same time the blood picks up wastes, carbon dioxide, and heat.

Cardiology is the branch of medicine dealing with disorders of the heart and blood vessels. To simulate an ECG/arrhythmia patterns, the literature on heart, about the structure (anatomy) of the heart which is the source of ECG, Conduction System of the heart i.e. how the impulse is generated and conducted to various parts of the heart and body and how the electrocardiogram (ECG) is generated is needed to understand first. This also describes about the details of the ECG waves, their amplitude, timings and abnormalities, and also gives the information about the arrhythmias, What are these, their classification, description, features, symptoms and causes about major life threatening arrhythmias which is of clinical significance to the cardiologist. This is discussed in the following sections.

2.2 THE HEART

The heart is a four chambered pump with two atria for collection of blood and two ventricles for pumping out of blood. Coordinated electrical events and a specialized

conduction system intrinsic and unique to the heart play major roles in the rhythmic contractile activity of the heart.

2.2.1 LOCATION AND SIZE OF THE HEART

The hollow, cone-shaped heart [10] is relatively small, about the same size as a person's closed fist, and weighs only about 300 gm in an adult [10]. The heart contains four chambers, to be described shortly: two upper atria and two lower ventricles. It rests on the diaphragm, near the middle of the thoracic cavity in a space called the mediastinum, which extends from the sternum to the vertebral column between the lungs. About two-thirds of the mass of the heart lies to the left of the body's midline. The heart is about 12 cm (5 in.) long, 9 cm (3 & ½ in.) wide at its broadest point, and 6 cm (2 & ½ in.) thick [10]. The pointed end of the heart, the apex, is formed by the tip of the left ventricle and tilts obliquely toward the left hip. Opposite the apex, the wide upper and posterior margin of the heart is called the base, so named because it is broad and rather flat, like the base of a pyramid, The base of the heart is formed by the atria, mostly the left atrium.

2.2.2 BASIC ANATOMY OF THE HEART

* Epicardium
* Pericardium
* Myocardium
* Papillary muscle
* Endocardium
* Coronary Circulation
* Heart valves

Figure 2.1 shows structure of the heart.

Pericardium

The pericardium [10] is a triple-layered bag that surrounds and protects the heart. It confines the heart to its position in the mediastinum, yet allows it sufficient freedom of movement for vigorous and rapid contraction. The pericardium consists of two principal portions : the fibrous pericardium and the serous pericardium. The outer fibrous pericardium is a tough, inelastic, fibrous connective tissue. It resembles a bag that rests on

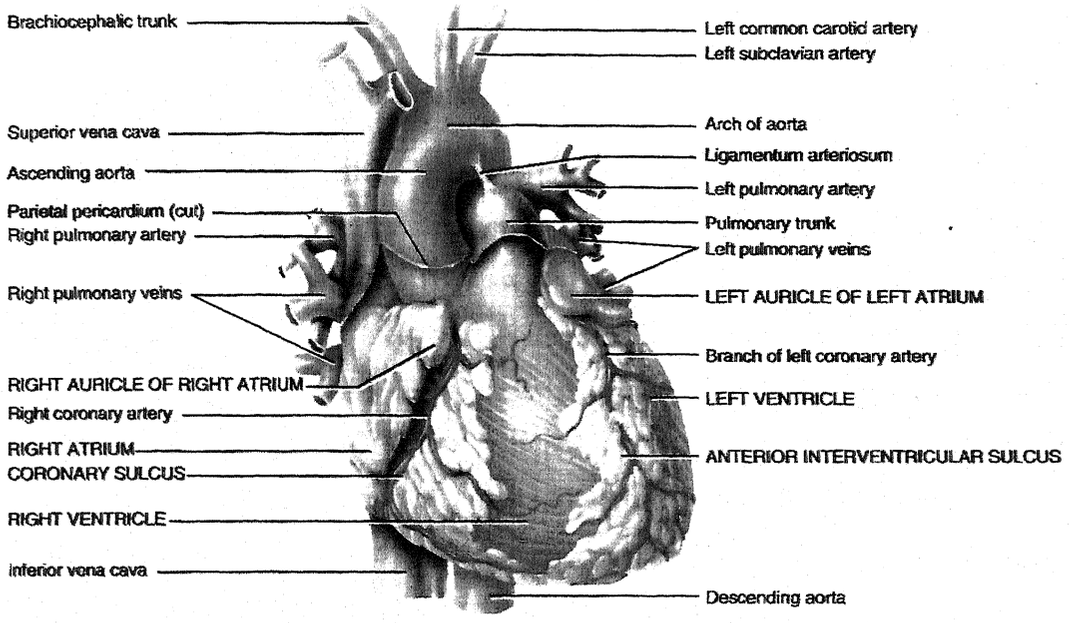


Figure 2.1 : Structure of the heart.

and attaches to the diaphragm with its open end fused to the connective tissues of the blood vessels entering and leaving the heart. Its lateral surfaces lie against the parietal pleurae, the outer coverings of the lungs. The fibrous pericardium prevents overstretching of the heart, provides protection and anchors the heart in the mediastinum.

The inner serous pericardium is a thinner, more delicate membrane that forms a double layer around the heart. The outer parietal layer of the serous pericardium is fused to the fibrous pericardium. The inner visceral layer of the serous pericardium, also called the epicardium adheres tightly to the muscle of the heart. Between the parietal and visceral layers of the serous pericardium is a thin film of serous fluid known as pericardial fluid. It is a slippery secretion of the pericardial cells and reduces friction between the membranes as the heart moves. The space that houses the pericardial fluid is called the pericardial cavity [10].

Heart Wall

Three layers from the wall of the heart the epicardium (external layer), myocardium (middle layer), and endocardium (inner layer). The outermost epicardium

also called the visceral layer of the serous pericardium is the thin, transparent outer layer of the wall. It is composed of mesothelium and delicate connective tissue that imparts a smooth, slippery texture to the outermost surface of the heart.

The middle myocardium [10], which is cardiac muscle tissue, makes up the bulk of the heart and is responsible for its pumping action. Cardiac muscle fibers (cells) are involuntary, striated and branched. They swirl diagonally around the heart in interlacing bundles and form two large networks- one atrial and one ventricular. Each fiber physically contacts neighbouring fibers in its network by transverse thickenings of the sarcolemma called intercalated discs. Within the discs are gap junctions (electrical synapses) that allow muscle action potentials to spread from one fiber to another. As a result, the whole atrial network contracts as another. The intercalated discs also contain desmosomes, which act as reinforcing spot welds. They prevent adjacent cardiac fibers pulling apart during their vigorous contractions.

The innermost endocardium [10] is a thin layer of endothelium overlying a thin layer of connective tissue. It provides a smooth lining for the inside of the heart and covers the valves of the heart. The endocardium is continuous with the endothelial lining of the large blood vessels associated with the heart and the rest of the cardiovascular system.

Chambers of the heart

The interior of the heart is divided into four compartments called chambers [1, 10] that receive the circulating blood. The two superior chambers are called the right atrium and left atrium. Each atrium has an appendage called an auricle [10], so named because its shape resembles a dog's ear, The auricles increase the volume of the atria. The two inferior chambers are the right ventricle and left ventricle.

Connective tissue separates the muscle tissue of the atria from that of the ventricles and effectively divides the myocardium into separate atrial and ventricular muscle masses. Externally, a groove known as the coronary sulcus and posterior interventricular sulcus separate the right and left ventricles externally. Sulci contain coronary blood vessels and a variable amount of fat.

A partition called the interatrial septum separates the atria. A prominent feature of this septum is an oval depression, the fossa ovalis. This was the site of the foramen ovale, an opening in the interatrial septum of the fetal heart. The irregular surface of ridges and folds of the myocardium covered by endocardium in the ventricles is known as the trabeculae carneae. A wall known as the interventricular septum [10] separates the two ventricles. The thickness of the walls of the four chambers varies according to their functions. The atria are thin-walled [10, 11] because they only have to deliver blood into the ventricles. Although the right and left sides of the heart act as two separate pumps, the left side has much larger workload. Whereas the right ventricle pumps blood only to the lungs (pulmonary circulation), the left ventricle pumps blood to all other parts of the body (systemic circulation). Thus the left ventricle must work harder than the right ventricle to maintain the same rate of blood flow. The anatomy of the two ventricles confirms this functional difference : the muscular wall of the left ventricle is two to four times as thick as the wall of the right ventricle.

Blood flow through the heart

The right atrium receives deoxygenated blood (blood that has given up some of its oxygen to cells) from various parts of the body through three veins. In general the superior vena cava (SVC) brings blood from most parts of the body superior to the heart, the inferior vena cava (IVC) brings blood from all parts of the body inferior to the diaphragm, and the coronary sinus drains blood from most of the vessels supplying the wall of the heart [10].

From the right atrium blood flows into the right ventricle, which pumps it to the lungs, starting in the pulmonary trunk. The pulmanory trunk divides into a right and left pulmonary artery, each of which carries blood to one lung. In the lungs, the blood releases carbon dioxide and takes on oxygen, This blood, called oxygenated blood, returns to the heart via four pulmonary veins that empty into the left atrium. The blood then passes into the left ventricle, which pumps the blood into the ascending aorta. From here the blood flows into the coronary arteries, which carry the blood to the heart, arch of the aorta, thoracic aorta and abdominal aorta [10,12]. The aorta and its branches carry the blood throughout the systemic circulation.

Valves of the heart

As each chamber of the heart contracts, it pushes a portion of blood into a ventricle or out of the heart through an artery. To prevent backflow of blood [10, 12], the heart has valves. These structures are composed of dense connective tissue covered by endocardium. Valves open and close in response to pressure changes as the heart contracts and relaxes [10, 12, 13].

Atrioventricular Valves

Atrioventricular (AV) valves [10] lie between the atria and ventricles. The right AV valve between the right atrium and right ventricle is also called the tricuspid valve because it consists of three cups (flaps). The left AV valve between the left atrium and left ventricle has two cups and is called the bicuspid [10].

Semilunar Valves

Both arteries that emerge from the heart have a valve that prevents blood from Hawing backward into the heart. These are the semilunar (SL) valves [10]. The pulmonary semilunar valve lies in the opening where the pulmonary trunk leaves the right ventricle, The aortic semilunar valve is situated at the opening between the left ventricle and the aorta.

Both valves consist of three semilunar (half-moon. or crescent-shaped) cusps, Each cusp is attached by its convex margin to the artery wall, The free borders of the cusps curve outward and project into the opening inside the blood vessel. Like the atrioventricular valves, the semilunar valves permit blood to flow in one direction only in this case, the flow is from the ventricles into the arteries.

Heart Blood Supply

The wall of the heart has its own blood vessels. Nutrients could not possibly diffuse through all the layers of cells that make up the heart tissue. The flow of blood through the many vessels that pierce the myocardium is called the coronary (cardiac) circulation [10]. The arteries of the heart encircle it like a crown encircles the head (corona = crown).

Coronary Arteries

Two coronary arteries as the right and left coronary arteries, branch from the ascending aorta. The left coronary artery courses under the left auricle and divides into the anterior interventricular and circumflex branches. The anterior interventricular branch or left anterior descending (LAD) artery is in the anterior interventricular and supply oxygenated blood to the walls of both ventricles. The circumflex branch lies in the coronary sulcus and distributes oxygenated blood to the walls of the left ventricles and left atrium.

The right coronary artery supplies small branches to the right atrium. It continues under the right auricle and divides into the posterior interventricular and marginal branches. The posterior interventricular branch follows the posterior interventricular sulcus and supplies the walls of the two ventricles with oxygenated blood. The marginal branch in the coronary sulcus transports oxygenated blood to the myocardium of the right ventricle. The left ventricle receives the most abundant blood supply because of the enormous work it must do.

Most parts of the body receive branches from more than one artery, and where two or more arteries supply the same region, they usually connect. The connections, called anastomoses provide alternate routes for blood to reach a particular organ or tissue. The myocardium contains many anastomoses, connecting branches of one coronary artery or extending between branches of different coronary arteries. Heart muscle can remain alive if it receives as little as 10 to 15% of its normal blood supply.

Coronary Veins

As blood passes through the coronary circulation, it delivers oxygen and nutrients and collects carbon dioxide and wastes. It then drains into a large vein on the posterior surface of the heart called the coronary sinus, which empties into the right atrium. A vascular sinus is a vein with a thin wall that has no smooth muscle to alter its diameter. The principal tributaries carrying blood into the coronary sinus are the great cardiac vein, which drains the anterior aspect of the heart, and the middle cardiac vein, which drains the posterior aspect of the heart.

2.3 CONDUCTION SYSTEM

An inherent and rhythmical electrical activity [13] is the force behind the heart's continuous beating. Certain cardiac muscles [13, 14, 15]cells repeatedly fire spontaneous impulses (action potentials) that then trigger heart contractions. Therefore a heart that has been completely removed from the body, for example, to be transplanted into another person will continue to beat even though all its nerves have been cut. Signals from the autonomic nervous system and hormones, such as epinephrine, in the blood do modify the heartbeat [16, 17], but they do not establish the fundamental rhythm.

2.3.1 AUTORHYTHMIC CELLS

During embryonic development, a small fraction (about 1 %) of the cardiac muscle fibers become autorhythmic (self-excitable), that is, able to repeatedly and rhythmically generate impulses [15, 18, 19]. The autorhythmic fibers have two important functions. They act as a pacemaker [18] setting the rhythm [16] for the entire heart, and they form the conduction system, the route for conducting impulses throughout the heart muscles. The conduction system assures that cardiac chambers contract in a coordinated manner, which makes the heart an effective pump. The components [14] of the conduction system are: the sinoatrial (SA) node, the atrioventricular (AV) node. The atrioventricular (AV) bundle (bundle of His), the right and left bundle branches, and the conduction myofibers (Purkinje fibers).

Normally, cardiac excitation begins in the sinoatrial (SA) node located in the right atrial wall just below the opening of the superior vena cava. Each SA node impulse travels throughout the heart via the conduction system and the gap junctions in the intercalated discs, in the wake of the impulse first the atria contract and then the ventricles contract.

The cardiac impulse [18] spreads from the SA node throughout the atrial fibers and down to the atrioventricular (AV) node located in the septum between the two atria. From the AV node the impulse enters the atrioventricular (AV) bundle (bundle of His), the only electrical connection between the atria and the ventricles. (Elsewhere, fibrous rings and sheets of connective tissue act as electrical insulation between the atria and ventricles.) After traveling along the AV bundle the impulse then enters both the right, and left bundle branches, that course through the interventricular septum toward the apex of the heart. Finally, large-diameter conduction myofibers (Purkinje fibers) rapidly conduct the impulse into the mass of ventricular muscle tissue.

Autorhythmic fibers in the SA node spontaneously initiate action potentials [10, 14, 18] 60 to 100 times per minute, faster than any other region. As a result, action potentials from the SA node spread to other areas of the conduction system, stimulating them before they are able to generate an impulse at their own slower rate. Thus the normal pacemaker of the heart is the SA node.

Sometimes a site other than the SA node becomes the pacemaker because it develops abnormal self-excitability. Such a site is called an ectopic pacemaker or ectopic focus. The ectopic focus may operate only occasionally, producing extra beats, or it may pace the heart for some period of time. Triggers of ectopic activity include caffeine and nicotine, electrolyte imbalances, hypoxia, and toxic reactions to drugs such as digitalis [1].

2.3.2 TIMING OF ATRIAL AND VENTRICULAR EXCITATION

From The SA node the cardiac impulse travels (throughout the atrial muscle and down to the AV node in about 0.05 sec (50 milliseconds or msec). The impulse slows considerably at the AV node because the fibers there have much smaller diameters. The resulting 0.1 sec (100 msec) delay has an advantage. It gives the atria, time to complete their contraction and add to the volume of blood in the ventricles before ventricular contraction begins. After the cardiac impulse enters the AV bundle conduction again is rapid. The entire ventricular myocardium undergoes depolarization [10] (loss and then reversal of polarization) about 0.15 to 0.2 sec (150 to 200 msec) after the impulse arises in the SA node.

If the SA node becomes diseased or damaged, the slower AV node fibers can pick up the pacemaking chores. With pacing by the AV node, heart rate ranges between 40 and 50 beats/min. If the activity of both nodes is suppressed, the heartbeat may still be maintained by autorhythmic fibres in the ventricles-the AV bundle, a bundle branch, or conduction myofibers. However, these fibers fire impulses very slowly, only about 20 to 40 times per minute. At such an abnormally low heart rate blood flow to the brain in is inadequate. In patients with such a conditions normal heart rhythm can be restored and maintained with an artificial pacemaker, a device that sends out small electrical currents that stimulate the heart. Many of the newer pacemakers, called activity-adjusted pacemakers automatically speed up the heartbeat during exercise

2.3.3 PHYSIOLOGY OF CARDIAC MUSCLE CONTRACTION

The impulse initiated by the SA node travels along the conduction system [14, 15] and spreads out to excite the 'working' atrial and ventricular muscle fibers, which are called contractile fibers. The contractile fibers have a resting membrane potential [10, 16]close to -90 mV, when they are brought to threshold by excitation in neighbouring fibers. Certain sodium ion channels opens very rapidly; these are called voltage-gated fast Na^+ channels. This increase in membrane permeability allows an inflow of Na^- down its concentration gradient and produces a rapid depolarization. Refer Figure 2.2 for impulse (Action Potenial) in a ventricular Contractile Fiber.

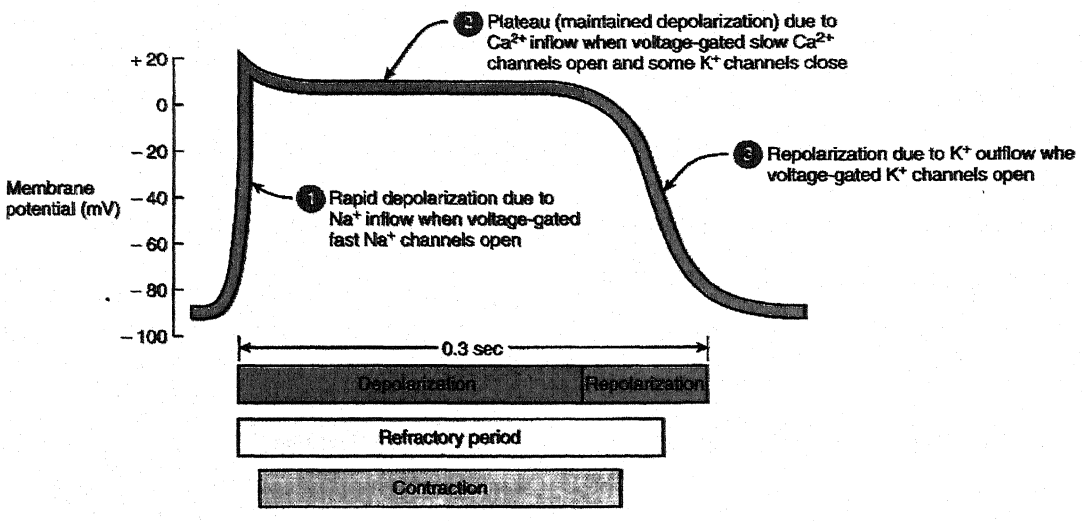


Figure 2.2 : Impulse (Action Potential) in a ventricular Contractile Fiber.

During the next phase, called the plateau, voltage-gated slow Ca^{2+} channels open, allowing calcium ions enter the cytosol. Some Ca^{2+} passes through the sarcolemma (plasma membrane) from the extra cellular fluid (which has a higher Ca^{2+} concentration) while other calcium pours out of the sarcoplasmic reticulum within the fiber. The combined buildup of Na^+ and Ca^{2+} in the cytosol maintains the depolarization for about 0.25 sec (250 msec). By comparison, depolarization in a neuron or skeletal muscles fiber lasts about 1 msec.

The next steps are similar in skeletal and cardiac muscle fibers. Ca^{2-} binds to troponin, which allows the actin and myosin filaments to begin sliding past one another,

and tension starts to develop. Substances that alter the movement of Ca^{2+} through slow Ca^{2+} channels influence the strength of heart contractions Epinephrine, for e.g., increases contraction force by enhancing Ca^{2+} inflow. Certain drugs, appropriately called calcium channel blockers such as verapamil reduce Ca^{2+} inflow and diminish the strength of the heartbeat.

The repolarization (recovery of resting membrane potential) phase of the impulse in a cardiac muscle fiber resembles repolarization in other excitable tissues after a delay (which is particularly prolonged in cardiac Muscle), voltage-gated K^+ channels open, and potassium ions diffuse out along their concentration gradient. At the same time, the Na^+ and Ca^{2+} channels are closing which slows and then almost stops further inflow of these two ions. As more K^+, leaves the fiber and fewer Na^+ and Ca^{2+} enter the negative resting membrane potential (-90 mV) is restored and the muscle fiber relaxes.

In muscle the refractory period is the time interval when a second contraction cannot be triggered. The refractory period of a cardiac fiber is longer than the contraction itself. As a result, another contraction cannot begin until relaxation is well underway and tetanus (maintained contraction) cannot occur. The pumping function of ventricles depends on alternating contraction, when they eject blood and relaxation, when they refill. If tetanus could occur blood flow would stop.

2.3.4 CARDIAC ACTION POTENTIAL

The cardiac action potential [1, 10] is the electrical activity of the individual cells of the electrical conduction system of the heart.

The cardiac action potential differs significantly in different portions of the heart. This differentiation of the action potentials allows the different electrical characteristics of the different portions of the heart. For instance, the specialized conduction tissue of the heart has the special property of depolarizing without any external influence. This is known as automaticity.

The electrical activity of the specialized conduction tissues are not apparent on the surface electrocardiogram (ECG) [13]. This is due to the relatively small mass of these tissues compared to the myocardium (muscle of the heart).

Resting membrane potential

The resting membrane potential [10] is the difference in ionic charge across the membrane of the cell during phase 4 of the action potential. Table 2.1 shows intracellular and extracellular ion concentrations.

Table 2.1 Intracellular and extracellular ion concentrations

Ion	Extracellular concentration (mM)	Intracellular concentration	Ratio of extracellular to intracellular concentration
Na^+	145	15 mmol/L	9.7
K^+	4	150 mmol/L	0.027
Cl^-	120	5-30 mmol/L	4-24
Ca^{2+}	2	10-7 mml/L	$2x10^4$
Although Intracellular Ca2+ content is about 2 mM, most of this is bound or sequestered in intracellular organelles (mitochondria and sarcoplasmic reticulum)			

The normal resting membrane potential in the ventricular myocardium is about -85 to -95 mV. This potential is determined by the selective permeability of the cell membrane to various ions. The resting membrane potential is permeable to K^+, and is relatively impermeable to other ions. The resting membrane potential is therefore determined by the K^+ gradient across the cell membrane (the reversal potential for K^+). The maintenance of this electrical gradient is due to various ion pumps and exchange mechanisms, including the Na^+ - K^+ ion exchange pump and the Na^+ - Ca^{2+} exchange mechanism.

Intracellularly (within the cell), K^+ is the principle cation, and phosphate and the conjugate bases of organic acids are the dominant anions. Extracellularly (outside the cell), Na^+ and Cl^- predominate.

Phases of the cardiac action potential

The standard model used to understand the cardiac action potential is the action potential of the ventricular myocyte. The action potential has 5 phases (numbered 0 - 4).

Phase 4 is the resting membrane potential, and describes the membrane potential when the cell is not being stimulated.

Once the cell is electrically stimulated (typically by an electric current from an adjacent cell), it begins a sequence of actions involving the influx and eflux of multiple cations and anions that together produce the action potential of the cell, propogating the electrical stimulation to the cells that lie adjacent to it. In this fashion, an electrical stimulation is conducted from one cell to all the cells that are adjacent to it, to all the cells of the heart. Refer figure 2.3 for Cardiac Action Potential Phases.

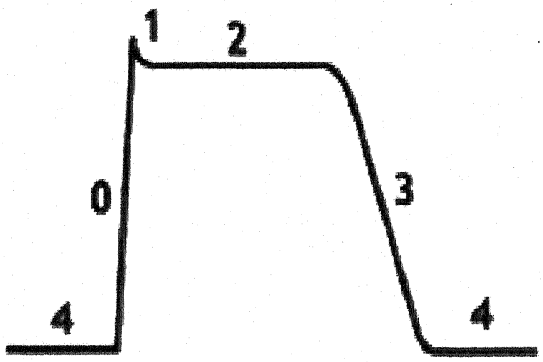


Figure 2.3 : Cardiac Action Potential Phases

Phase 4

Phase 4 is the resting membrane potential. This is the period that the cell remains in until it is stimulated by an external electrical stimulus (typically an adjacent cell). This phase of the action potential is associated with diastole [12, 13] of the chamber of the heart.

Certain cells of the heart have the ability to undergo spontaneous depolarization, in which an action potential is generated without any influence from nearby cells. This is also known as automaticity. The cells that can undergo spontaneous depolarization the fastest are the primary pacemaker cells of the heart, and set the heart rate [16]. Usually, these are cells in the SA node of the heart. Electrical activity that originates from the SA node is propagated to the rest of the heart. The fastest conduction of the electrical activity is via the electrical conduction system [14] of the heart.

In cases of heart block [20, 21, 22], in which the activity of the primary

pacemaker does not propagate to the rest of the heart, a latent pacemaker (also known as an escape pacemaker) will undergo spontaneous depolarization and create an action potential.

The mechanism of automaticity is still unclear. Depolarization of SA and AV nodal cells largely depend on a net increase in intracellular positive charge [23]. Mechanisms include a decrease in the net K^+ outward flow, and a time-dependent increase in flow of Na^+ and Ca^{2+} ions.

Phase 0

Phase 0 is known as the rapid depolarization phase. The slope of phase 0 is determined by the maximum rate of depolarization of the cell and is known as Vmax. This phase is due to opening of the fast Na^+ channels and the subsequent rapid increase in the membrane conductance to Na^+ (g_{Na}) and a rapid influx of ionic current in the form of Na^+ ions (I_{Na}) into the cell.)

The ability of the cell to open the fast Na^+ channels during phase 0 is related to the membrane potential at the moment of excitation. If the membrane potential is at its baseline (about -85 mV), all the fast Na^+ channels are closed, and excitation will open them all, causing a large influx of Na^+ ions. If, however, the membrane potential is less negative, some of the fast Na^+ channels will be opened earlier, causing a lesser response to excitation of the cell membrane and a lower Vmax.

The maximal fast inward Na^+ current is generated when the membrane potential is at the normal resting potential (-85 to -95 mV). If the resting membrane potential is reduced to a low enough level, the increase in fast inward Na^+ current may be inadequate to produce a response, making the fiber unexcitable

The fast Na^+ channel

The fast sodium channel is made up of two gates, the m gate and the h gate. It is the interaction of these two gates that allows Na^+ to enter the cell through this channel. In the resting state, the m gate is closed and the h gate is open. Upon electrical stimulation of the cell, the m gate opens quickly while simultaneously the h gate closes slowly. For a brief period of time, both gates are open and Na^+ can enter the cell across the electrochemical gradient.

Phase 1

Phase 1 of the action potential is due to closure of the fast Na^+ channels. The transient net outward current is due to the movement of K^+ and Cl^- ions.

Phase 0 and 1 together correspond to the R and S waves of the EGG.

Phase 2 of the action potential corresponds to the ST segment of the EGG.

Phase 3

During phase 3 of the action potential, the K^+ channel is still open, allowing more K^+ to leave the cell and accumulate in the extracellular space. This net loss of positive charge causes the cell to repolarize. The k^+ channels close when the membrane potential is restored to about -40 to -45 mV.

Phase 3 of the action potential corresponds to the T wave [24] on the EGG.

2.3.5 PACEMAKER TISSUES

Certain tissues in the heart [10, 12, 13], concerned with the initiation (generation of impulses) and propagation (conduction) of the heart beat, are called pacemaker tissues. They include:

1. Sinu or Sino Atrial Node (SAN)
2. Atrio-Ventricular Node (AVN)
3. Atrio-Ventricular Bundle or Bundle of His
4. Purkinje fibers that is ramification of Bundle of His

Sinu Atrial Node (SAN)

I. Location: on the posterior aspect of heart [10, 12] at the junction of the superior venacava (SVC) with right atrium (RA)(free b order of the RA appendix).

II. Dimensions: Length-15mm; Width-2mm and Thickness-1mm.

III. Structure: more embryonal in character i.e. cell outline ill defined; highly vascular; consists of thin, elongated muscle fibers (approx. $1/3^{rd}$ the size of heart muscle fibers); rich in glycogen and mitochondria, fusiform in shape with logitudnal striations. These are called P-cells or pacemaker cells. These fibers normally can generate and discharge impulses more rapidly than any other

pacemaker tissue and their rate of discharge determines the rate at which the heart beats. Because cardiac myocytes, like all nerve cells, have refractory periods following contraction during which additional contractions cannot be triggered, their pacemaker potential is overridden by the sinoatrial node. Cells in the SA node will naturally discharge (create action potentials) at about 70-80 times/minute. Because the sinoatrial node is responsible for the rest of the heart's electrical activity. SAN is called the Cardiac Pacemaker [10, 12, 13].

IV. Innervation: It develops from structures on the right side of the embryo. That is why, in adults, SAN is innervated by right vagus nerve. It also receives sympathetic nerve fibers predominantly of right side from the cervical sympathetic ganglia via the cardiac nerves. This makes the SA node susceptible to autonomic influences. Stimulation of the vagus nerve causes decrease in the SA node rate (thereby causing decrease in the heart rate). Stimulation via sympathetic fibers causes increase in the SA node rate (thereby increasing the heart rate).

In the majority of patients, the SA node receives blood from the right coronary artery, meaning that a myocardial infarction occluding it will cause ischaemia in the SA node unless there is a sufficiently good anastomosis from the left coronary artery. If not, death of the affected cells will stop the SA node from triggering the heartbeat.

Atrio ventricular node (AVN)

If the SA node doesn't function, or the impulse generated in the SA node is blocked before it travels down the electrical conduction system, a group of cells further down the heart will become the heart's pacemaker. These cells form the atrioventricular node (AV node), which is an area between the atria and ventricles, within the atrial septum.

I. Location: posteriorly on right side of the interatrial septum near the opening of coronary sinus.

II. Structure: same as that of SAN.

III. Innervation: It is a left sided structure of the embryo. Therefore, in adults,it is innervated by left vagus nerve; also receives sympathetic nerve supply simply from left side.

Atrio-ventricular bundle or the bundle of His

I. It take origin from AVN and then runs upwards to the posterior margin of the membranous inter-ventricular septum and then forwards below it, ensheathed and isolated in the canal. At the anterior part of the membranous inter-ventricular septum the bundle divides into a left and right branch.

II. The left branch pierces the membrane and then lies on the upper border of the muscular septum to divide into an anterior fascicle and a posterior fascicle. The right branch passes down the right side of the septum.

III. Both branches divide repeatedly to form a network of fibers lying subendocardially in the ventricles.

Purkinje fibers

I. Takes origin from terminal divisions of right and left branch of the bundle of His to penetrate the ventricular wall.

II. These fibers [13] are somewhat larger and thicker than the cardiac muscle fibers; length: 10-46μm and diameter 70-80 μm with indistinct cell outlines and granular central cytoplasm containing several nuclei; the peripheral cytoplasm [17] contains myofibrillae and is rich in glycogen content.

III. Because of the large diameter they transmit the impulse at a fast velocity of 4mts/sec as compared to other conducting tissue. This allows almost immediate transmission of the cardiac impulse throughout the entire ventricular system. Table 2.2 gives conduction speed in Cardiac Tissue.

Table 2.2 : Conduction speed in Cardiac Tissue.

Cardiac Tissue	Conduction Speed (mts/sec)
I. Sino Atrial Node	0.05
II Atrial Pathways	1.0
III Atrio-ventricular node	0.05
IV Bundle of His	1.0
V Purkinje system	4.0
VI Ventricular muscles	1.0
VII Atrial muscles	1.0

Refer Figure 2.4 for Conduction System of the Heart. Pacemaker activity at the SA node reaches the AV node by cell – to –cell atrial conduction. The conduction system carries the impulse from the AV node to the working ventricular muscle by traversing the common bundle of His, the right or left bundle branch, and the Purkinje network. RA, right atria; LA, left atria; RV, right ventricle ; LV, left ventricle.

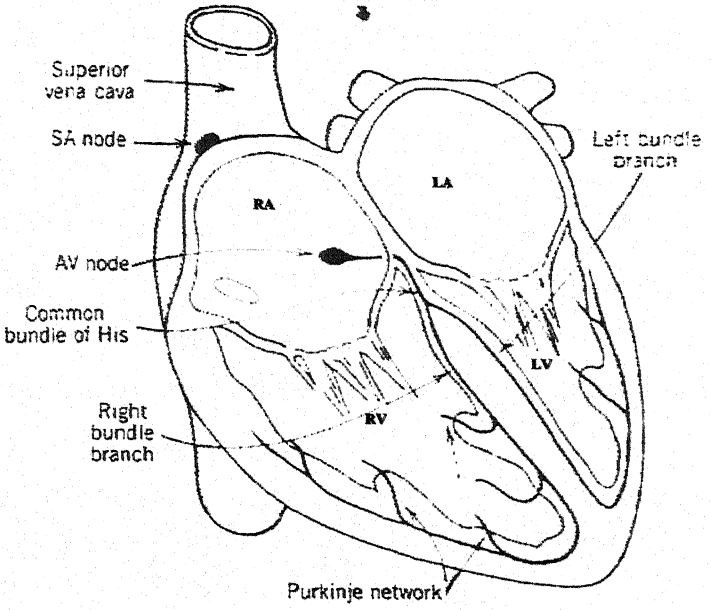


Figure 2.4 : Conduction System of the Heart

Pacemaker cells are located in the region of the sinoatrial (SA) node, which is a bit of tissue the size of a pencil tip located at the site of entry of the descending vena cava.

Repetitive activity is initiated at this point and propagates to adjoining atrial tissue by means of the local-circuit (action) currents. The flow of this current from active to inactive neighboring cells is facilitated by the presence of low-resistance intercellular structures. Activation proceeds in this way from cell to cell until the entire right and then left atria are activated. (The nature of the intercellular structures in both atrial and ventricular tissue will be described subsequently in this section.) Because the atria and ventricles are separated by fibrous tissue, direct propagation from the atria to ventricles cannot occur. Instead activation must follow a path that starts in the atria at the atrioventricular (AV) node and proceeds through the common and then right and left bundles of His, to the terminal Purkinje fibers which arborize and invaginate the endocardial ventricular tissue. The initial part of this path involves slow conduction in the A V junction. Since electrical activation of cardiac muscle initiates the successive mechanical contraction, this results in a delay in ventricular activation and contraction that is beneficial. Since it allows for completion of atrial contraction. Once the electrical impulse reaches the bundles of His, conduction is very rapid, resulting in the initiation of ventricular activation over a wide region. The subsequent cell-to-cell propagation is consequently sequenced and coordinated, resulting in a mechanical contraction that is similarly synchronized and efficient.

Activation of Ventricular musculature, initiated at many endocardial sites by the conduction system, proceeds generally from endocardium to epicardium through cell-to-cell conduction. Such conduction is made possible by low-resistance intercellular junctions, which permit the flow of a sufficient fraction of prejunctional action current to the post junctional cell to result in activation of the latter. Specific low-resistance hexagonal structures called connexons have been identified through freeze fracture electron microscopy; the connexons are organized in Hexagonal arrays that comprise the gap junctions. It is a central tube of the connexon that is believed to constitute an intercellular channel for the movement of ions and small molecules. The connexons span a gap between adjoining cells that is narrowed to 30 μm from a normal 200 μm intercellular space. If one thinks of two end-toend cells in the shape of successive tank cars, the connexons represent a number of interconnecting pipes providing for interunit cytoplasmic continuity. In this model one can understand how action currents arising in a prejunctional cell can flow, relatively easily, into the post junctional cell and then

outward across the membrane. In so doing, a depolarizing transmembrane potential is produced in the post junctional cell, which will eventually reach threshold in much the same way as this occurs at a downstream site in a uniform continuous (single) cylindrical cell.

Cardiac ventricular tissue when viewed on a larger scale is seen to be composed of bands of fibers that spiral around the central cavity, If passive gross electrical conductivity measurements are made at a specific site, conductivity along the fiber axis will be found to exceed that transverse to the fibers by a factor of 4 or so. One consequence is that propagation of an impulse in the fiber direction will proceed at a velocity that is around two times greater than that in the direction normal to fiber, These anisotropies play an important role in determining activation patterns and source strength and configuration. On the other hand for the most part, principal effects can be discerned if one assume isotropic behaviour. Such an assumption implies that all cells connect equally well to all neighbors and that electrical activity initiated at a point would propagate outward as a spherical wave. The heart muscle [15] is referred to as syncytial reflecting an electrically excitable continuum.

2.3.6 (SINGLE-FIBER) ELECTROPHYSIOLOGICAL PRINCIPLES

Activation

Nerves and muscles have the properties of electrical excitability. When stimulated the result is a cyclic movement of ions into and out of each activated cell, and this is accompanied by changes in transmembrane potential. The latter constitutes an electrical impulse that is seen to propagate from the site of excitation to adjoining regions.

This behaviour arises basically from the nature of the excitable membrane that bounds each cell. This membrane contains active elements (pumps), which establish and maintain an ionic intracellular composition that is different from the extracellular milieu. The intracellular space is high in potassium and low in sodium and chloride while the extracellular medium, conversely, has a low potassium concentration but high sodium and chloride concentrations. The additional membrane property that accounts for excitability is its selective permeability. At rest it is high for potassium so that the outward diffusion of this ion (alone) causes the membrane capacitance to develop a negative intracellular

charge (positive extracellularly). The consequence is a resting intracellular electrical potential that is negative relative to the extracellular space (taken as a reference), and this is in the order of 0.1 V.

If a stimulating current is applied such that a 'patch' of membrane is caused to have an increased transmembrane potential by a threshold amount (around l0-15 mV), the membranes permeabilities are caused to change through a cyclic process. The change is characterized by an initial rapid rise in sodium permeability to a dominant value and an equally rapid fall in this value toward its resting level; this is accompanied by a slower rise and fall in potassium permeability in the case of nerve and muscle (for cardiac muscle a series of permeability changes results in delayed recovery.

The concomitant transmembrane potential [12, 15] undergoes the phasic changes as described, and this constitutes an action potential. As noted previously the resting potential arises from potassium efflux due to diffusion and this ceases (i.e., a steady state is reached) when the inward electric field from the charge displacement equilibrates the outward diffusional force. This equilibrium can be evaluated through the application of electrical field theory (10) and diffusion theory, giving rise to the Nernst equation, which (for potassium) is

$$V_m = 25 \ln([K]_e/[K]_i) \qquad (1)$$

where Vm is the transmembrane potential in millivolts (the Nernst potential) $[K]_e$ is the extracellular potassium concentration, and $[K]_i$ is the intracellular potassium concentration. For typical values of $[K]_e = 4\mu$ mol/cm^3 and [K]i = 155 μ mol/cm^3, Vm = -91.4 mV is determined. The steady resting potential of a cardiac cell is approximated by the potassium Nernst potential. Activation (depolarization) arises when the transmembrane potential is increased say to -75 mV by abruptly passing a current that flows outward across the membrane. A consequence is a rapid increase in sodium permeability. The result is an incremental sodium influx (due to both the inward electric field and the inward sodium concentration gradient and this results in a further increase (algebraically) in transmembrane potential, and so on. The process is regenerative producing further increases in sodium permeability and transmembrane potential, and ends essentially when

the transmembrane potential reaches the sodium equilibrium (Nernst) potential. Typical values of intracellular and extracellular sodium concentrations in cardiac muscle are $[Na]_e = 145\mu$ mol/cm^3 $[Na]_i = 12\mu$ mol/cm^3,so that

$$V_m = 25\ln\left(\frac{145}{12}\right) = 62.3\text{mV} \qquad (2)$$

is the potential at the action potential peak; one notes a reversal in polarity at this time. Recovery consists mainly in the sodium and potassium permeabilities returning to their resting values and, consequently, a return of the transmembrane potential to its resting value. During the early period of recovery, the membrane is inexcitable (absolutely refractory) and, toward the close of recovery, can be excited, but requires an abnormally high stimulus (relatively refractory).

Propagation

If one end of a long cylindrical fiber [25] is excited by the application of a transthreshold stimulus, propagation of the action potential impulse to the opposite end is seen to occur. The extracellular pair of electrodes A, B is connected so that A is the cathode and B the anode of a stimulus current. The flow of current from B to A, which passes across the initially resting membrane in an inward direction at B and outward at A. The resting membrane can be characterized by a passive resistance and capacitance in parallel and the effect of the stimulating current will be an exponential change in transmembrane potential. From application of elementary electrical circuit concepts. This stimulating current is seen to depolarize the membrane at A and hyperpolarize it at B. Consider the IR drop in potential in the membrane resistance. If the stimulus current is large enough to exceed threshold at A. an action potential will be elicited at that site. Refer Figure 2. 5 for Stimulating Current flow following closure of switch corresponds to the passive circuit shown. Depolarisation takes place under the cathode A.

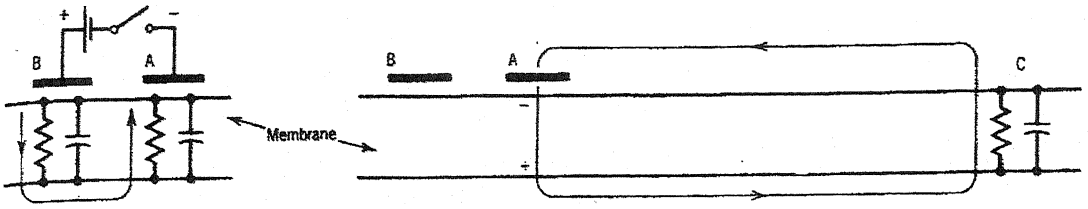


Figure 2.5 (a) Stimulating current flow following closure of switch corresponds to the passive circuit shown. Depolarization takes place under the cathode. A. (b) At a short time following (a), there is a rapid rise in sodium permeability at A, permitting a sodium influx and rise in transmembrane potential. The currents shown respond to the resulting differences in potential

The current flow pattern arising from initiation of excitation at A is depicted. The pattern results from the following considerations. Activation at A is associated with the rapid rise in sodium permeability at this site and results in the transmembrane potential approaching the sodium Nernst potential. Since the transmembrane potential is at rest in an adjoining region, such as C, currents must flow in the direction, as a result of the differences in potential. Since charge must be conserved, these comprise the closed current loops (called local-circuit currents) of particular importance is the outward current at site C. Since this region is subthreshold it behaves electrically as a resistance and capacitance in parallel as noted earlier. The transmembrane current, consequently, has two components- one a capacitive charging current and the second a resistive current (the latter being actually an ionic current). The transmembrane potential at C is depolarizing (since the intracellular potential is being made more positive). In real and normal physiological [12] preparations it turns out that the strength of the depolarization is always more than adequate to achieve activation. The result is that the pattern shifts to the right (C is now the activation site). The continued (sequential) operation of this mechanism accounts for propagation of the impulse along the fiber. The propagation rapidly reaches a uniform velocity so that if V m(t) is the temporal action potential, V m(z $-\theta t$) is its spacio temporal behavior, where θ is the velocity of propagation along the z axis. The field of electric currents associated with the propagating action potential is

referred to as action currents (or local-circuit currents), particularly when their role in supporting propagation is emphasized.

For a single fiber lying in an extensive volume conductor [25] and supporting a propagating action potential, the potential field in the extracellular medium is found to be small compared with intracellular values. An equivalent electrical network can be assigned this preparation reflecting the small extracellular potentials is the assignment of zero resistance to this region. For the intracellular space, its long and narrow character ensures axial current (only), and accordingly an axial ohmic resistance is assigned. The transmembrane path is shown nonspecifically. Since this may involve active as well as passive elements. Refer figure 2.6 for Linear Core conductor model [25] of an excitable fiber in an extensive extracellular space. The open boxes represent a parallel C_m Δz and $r_{m/\Delta z}$ if voltages are subthreshold or a more complex non linear representation if transthreshold. The intracellular potential is designated Φ_I

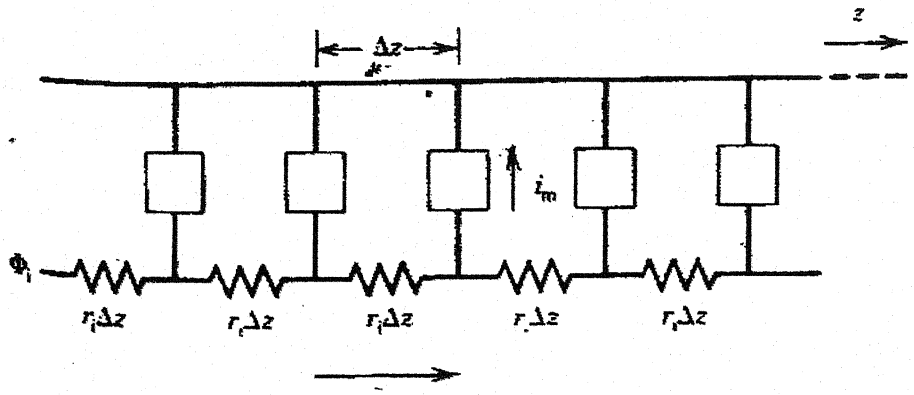


Figure 2.6 : Linear Core conductor model

Source/Field Relationship (Single Fibers)

Though the extracellular field is assumed to be small, it is not negligible. The transmembrane current enters the extracellular space and constitutes, thereby a potential field source. The strength of this current can be evaluated by applying Kirchhoff's circuit laws. Therefore

$$r_i I_i = -\partial\Phi_I / \partial z \qquad (3)$$

The above equation equates the intracellular potential [26] change in the z direction to the axial current Ii times the axial resistance per unit length ri, an application of Ohm's law. The transmembrane current per unit length i_m is related to the rate of decrease in intracellular axial current per unit length, since current must be conserved. Accordingly,

$$i_m = -\partial Ii/\partial z = (1/r_i)(\partial^2 \Phi i/ \partial z^2) \quad (4)$$

By defining the transmembrane potential Vm = Φi -Φe and since Φe≅0, ∴Φi≅Vm and

$$i_m = (1/r_i)(\partial^2 Vm/ \partial z^2) \quad (5)$$

Now an element of transmembrane current (i_m ∂z) constitutes a point current source within the extracellular volume conductor. If σ_e is the extracellular conductivity (assumed uniform) and the region is assumed to extend indefinitely, then the extracellular field of a point current source of strength $I_0 = i_m dz$ is

$$\Phi_e = I_0/(4\pi\sigma R) \quad (6)$$

where R is the distance from I_0 to an arbitrary field point. This formula can be verified calculating the extracellular electrical field $E = -\partial\Phi_e / \partial R = Io / (4\pi\sigma_e R^2)$ and the extracellular current destiny $J = \sigma_e E = Io / (4 \pi R^2)$, a value that conforms to the uniform radial outflow of current from a point source of magnitude I_0. The extracellular potential field from the entire fiber is found by super position of fields due to all point source elements (i_m ∂z) and applying in equation 6, this leads to

$$\Phi_e = (1 / (4\pi\sigma_e)) \int (i_m \partial z)/R \quad (7)$$

$$\Phi_e = (1/(4\pi\sigma_e r_i))\int (\partial^2 Vm/ \partial z^2)/R \quad (8)$$

In equation 8 , $\partial^2 Vm/ \partial z^2$ corresponds to an axial source density [26] so that $(\partial^2 Vm/ \partial z^2)$ dz behaves like a point source for extracellular fields. By applying this expression to active cardiac muscle which can be thought of as a collection of single fibers, which introduces the specific nature of such muscle.

2.3.7 CARDIAC SOURCES

Because of the syncytial nature of cardiac tissue one can consider the muscle cells as if they were arranged in an endocardial-epicardial direction (even though in fact they

lie transverse to this direction). But such an idealization permits to take into account its application to a multicellular structure to be applied for the evaluation of the cardiac sources of the electrocardiographic field. This is graphically illustrated in Figure 2.7. Refer Figure 2.7 for Evaluation of cardiac sources [15]. Phase 0 is shown spatially having been converted from its temporal waveform based on a rise time of around 1ms and a propagation velocity of 50 cm/s. Assuming an idealized plateau period following depolarization leads to the described source description of a leading positive source and trailing negative source [15]. Since it can be shown that the net positive and negative sources are equal in magnitude, the source arrangement constitute a double (or dipole) [24, 25] layer. It is clear infact that such a dipole layer arises along all isochrones (i.e., along all surfaces defining phase 0 cells) that exist at a particular time instant. As propagation proceeds in the usual endocardial-epicardial direction, double-layer sources accompany them and account for the space-time changing electrocardiographic field.

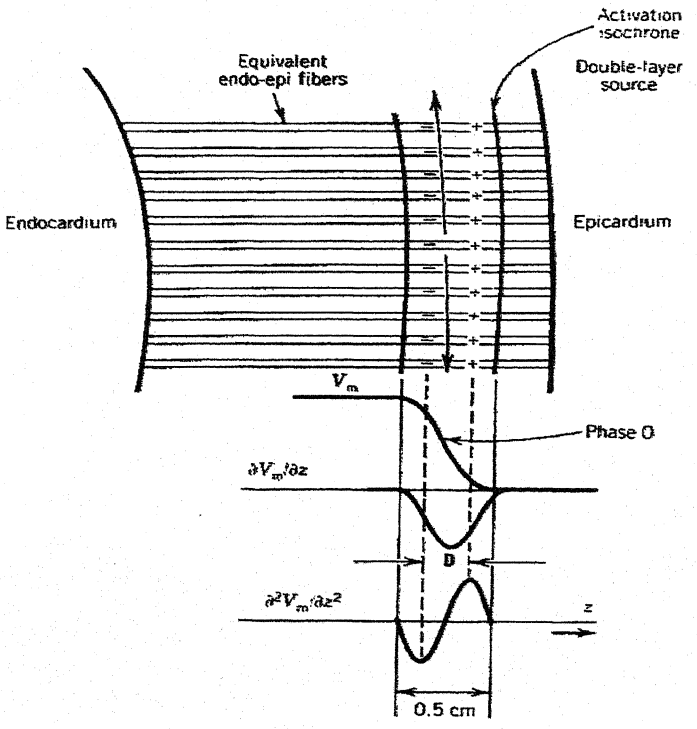


Figure 2.7 : Evaluation of cardiac sources

The strength of the dipole-layer source can be evaluated by the application. Apart from the factor 1/ri, the total positive source is found by integrating $\partial^2 Vm/\partial z^2$ from resting Vm to where $\partial Vm/\partial z$ peaks. This integration yields the value $(\partial Vm/\partial z)_{peak}$ since

$\partial Vm/\partial z = 0$, where Vm is resting. A similar magnitude is found for the total negative source. These magnitudes can be approximated as $(Vm_{peak} - Vm_{rest})/D$, where D is the separation of the positive and negative source. The dipole strength [25] involves multiplication of the single source magnitude by the source separation D. As a result the double-lay density τ, is proportional to $(Vm_{peak} - Vm_{rest})/ri$.

The preceding results, since they depend on what was derived for a single fiber source [15] in an unbounded volume conductor, must be modified to some extent to take into account the real (multicellular) cardiac tissue. The modifications affect the quantitative results, but leave the qualitative picture outlined here unchanged.

According to this (approximate) model the double layer source magnitude will be the same everywhere (a uniform, density) since it depends on $Vm_{peak} - Vm_{rest}$ which appears to be unvarying in experimental studies. The waveform of the electrocardiogram associated with ventricular depolarization is thus thought to arise from the changing shapes of the isochrones, hence double layers [24, 25], from the earliest moment of Purkinje activation to the time when the last bit of tissue has been reached and is activated by the advancing depolarization wave.

The field generated by a double layer can be thought of as the superposition of two component fields, one arising from the layer of positive sources and one from the layer of negative sources.

Denoting the sources layer density of each layer as W and their separation as D, then, as noted the dipole density strength $\tau = \mathbf{WD}$ (the bold type denotes a vector quantity and D is the displacement from negative to positive source along the normal to the isochrone). Keeping in mind that WdS behaves like a point source (10,11). Consequently,

$$\Phi = \frac{W}{4\pi\sigma} \int \left(\frac{1}{R^+} - \frac{1}{R^-} \right) dS \qquad (9)$$

where R^+ represents the source-field distance from elements, on the positive layer and R^- represents that from the negative layer. Since D is small compared with typical values of R^+ or R^- then Eq. can be rewritten as

$$\Phi = \frac{\tau}{4\pi\sigma} \int \nabla \left(\frac{1}{R} \right) .dS \qquad (10)$$

This result arises only because the double layer is assumed to be uniform and consequently its strength (density) can be removed from the integration. Although the sources actually occupy a region around 0.25 mm thick, the procedure has reduced this to an equivalent infinitesimal thickness and the double layer may be thought of as lying at some median position.

The preceding result can be expressed in terms of the net subtended solid angle, since an element of solid angle $d\Omega = \nabla(1/R).dS$. Consequently

$$\Phi = \frac{\tau}{4\pi\sigma} \int d\Omega \qquad (11)$$

where Ω is the solid angle subtended at the field point by the associated double layer. (If there is more than one double layer, as, e.g., one each may arise due to activation of the right ventricle, left ventricle, and septum, then it must be applied to each and the results superimposed). Since every activation wave with the same periphery has the same solid angle the field of each will be identical. Conversely, given a particular measured field there is no way to distinguish the responsible double-layer-source configuration beyond some particular solid angle, Finally, since a closed (unform) double layer has zero solid angle, it thereby generates zero potential.

2.3.8 EQUIVALENT SINGLE DIPOLE

The cardiac activation [24, 25] sources can be approximated as uniform double layers associated, at each instant of time, with the existing isochrones. A further simplification is possible if each elementary dipole is added to every other, vectorially, to form a single net dipole. Such an approximation, in effect, ignores the spatial distribution of the dipole elements. One could justify this simplification if the distance from source to field (i.e., from points in the heart to point on the torso) is large compared with the extent of the sources, which at most, is a linear heart dimension). Since precordial torso sites are as close or closer to the heart as the extent of the heart this is a questionable approximation. Nevertheless, experimental studies show that the procedure reflects first-order affects reasonably well.

The total dipole vector is assumed to have a fixed origin though a moving origin model has been explored and could be expected to reflect source behavior more accurately). Since activation double layers undergo a continuous smooth set of changes the net dipole can also be expected to change smoothly and continuously. The locus of the tip of this vector forms a closed loop in space and is called the vector loop; in clinical vectorcardiography it is specifically sought as a measure of the heart's electrical nature.

The representation of the electrical activity of the heart as a dipole fixed in location but free to change orientation and magnitude is obviously a great simplification in estimating the source generated by the heart. It also provides a great simplification in estimating the cardiac sources from measurements at the torso surface. (In principle, only three independent electrocardiographic potentials are needed to reconstruct the vector loop. Although the biophysical problem has been simplified, it retains features where one can often make a judicious guess from the behavior of the heart dipole of what may be happening at underlying isochronal double layers. The latter, in turn, can be directly related to the clinical status of the tissue.

Recovery

To this point almost all attention has been placed on the activation process in the heart. Cellular recovery depends on the time of activation, recovery of neighbours, and intrinsic membrane properties (temperature, metabolic activity. etc.). Recovery sources are generated, just as are activation sources, by the presence of spatial gradients of transmembrane potential. But while these could be inferred from the propagating activation wave (given the phase 0 morphology and propagation velocity) the many additional factors in recovery prevent an easy evaluation of these sources (recovery cannot he characterized is a propagating process). There is in fact no existing applicable model for recovery. On the other hand, most of cardiac diagnosis that is based on ECG waveform morphology utilizes only the activation period.

Although recovery sources are more difficult to evaluate than activation sources, it is clear that during recovery cardiac sources arise throughout the entire heart. Their source density magnitude is, however, small. Nevertheless, in the same way as described for activation, each dipole source element can be added to each other to obtain a net recovery heart vector. It, too, is an approximation since the spatial distribution of dipole

elements is ignored.) Since the underlying physical processes are uniform and continuous, the net recovery dipole has a continuous locus and defines a recovery (T-wave) vector loop. Even though the behavior of this vector loop is poorly understood on theoretical grounds, it has been found useful in empirical (statistical) cardiac diagnosis.

Potential Field Theory

The equation provides an explicit relationship between a double-layer source and the field it generates in a uniform homogeneous conducting medium. It is sometimes useful to regard the cardiac electrical sources as a volume distribution of dipoles described by a continuous function of position J_i. Obviously a system of double-layer sources congruent with the instantaneous set of isochrones is a special case of this more general formulation. The electrical potential field can be found from equation by recognizing that a dipole element τ dS and a dipole element $J_i dV$ must yield identical potential field expressions. Accordingly, for a uniform homogeneous conducting medium with conductivity σ,

$$\Phi = (1 / (4\pi\sigma)) \int J_i \nabla(1/R) dV \qquad (12)$$

If the Laplacian of both sides of equation 12 is taken, then it can be shown that

$$\nabla^2 \Phi = \nabla . J_i/\sigma \qquad (13)$$

which is a form of Poisson's equation. This result may be reached in a somewhat different way if we assume the presence of a continuous volume source density of current, I_v,. (I_v is analogous to charge density as a source of electrostatic flux.) The presence of the source I_v means that there must be a net outflow of current from any region containing I_v. More specifically, for a volume V bounded by a surface S the net outflow of current can be found by a surface integral of current density J and this must be equal to the net source (volume integral of source density I_v); thus

$$\int J.dS = \int I_v dV \qquad (14)$$

The outflow of current from a differential volume (outflow per unit volume) is evaluated by the divergence function, namely,∇.J, and this must equal I_v, according to equation 14 that is

$$\nabla . J = I_v \qquad (15)$$

Now, within the conducting medium. Ohm's law applies, namely,

$$J = \sigma E \tag{16}$$

where E is the electric field. Although all fields are time varying, at each instant of time they satisfy static equations to a condition described as quasi-static. In electrostatics, the electrical field may be derived as the (negative) gradient of a scalar potential function Φ. Consequently, we can rewrite equation 16 as

$$J = -\sigma \nabla \Phi \tag{17}$$

Now taking the divergence of eq 17. and substituting eq 15. Gives

$$\nabla^2 \Phi = -I_v / \sigma \tag{18}$$

which is an alternative expression of Poisson's equation for Φ. In fact, comparing the equations 13 and 18, it shows that

$$I_v = \nabla . J_i \tag{19}$$

which identifies a source description volume current (outflow) density with a source described as a volume dipole density. By duality with electrostatics, the solution of equation in integral form is

$$\Phi = (1/(4\pi\sigma)) \int (Iv/R) dV \tag{20}$$

2.4. GENERATION OF ECG AND ECG WAVES

Impulse conduction through the heart [10, 12, 13] generates electrical currents that can be detected at the surface of the body. A recording of the electrical changes that accompany each cardiac cycle (heartbeat) is called an electrocardiogram [43], abbreviated either ECG or EKG. The ECG is a composite of action potentials produced by all the heart muscle fibers during each heartbeat [16]. The instrument used to record the changes is an electrocardiograph. In clinical practice, the ECG [27] is recorded by placing electrodes on the arms and legs (the limb leads) and at six positions on the chest. As the person lies still the electrocardiograph amplifies the heart's electrical activity and produces 12 different tracings from different combinations of limb and chest leads. This takes about a minute. Each limb and chest electrode records slightly different electrical activity because it is in a different position relative to the heart. By comparing these

records [28] with one another and with normal records it is possible to determine if the conduction pathway is normal, if the heart is enlarged, and if certain regions are damaged. Refer Figure 2.8 for Normal ECG Waveform (Lead II).

In a typical Lead II record, three clearly recognizable waves accompany each heartbeat. The first called the P wave is a small upward wave. It represents atrial depolarization, which spreads from the SA node through both atria. About 0.1 sec after the P wave begins the atria contracts. The second wave, called the QRS complex begins as a downward deflection, continues as a large upright, triangular wave, and ends as a downward wave. The QRS complex represents the onset of ventricular depolarization [29, 30] the spread of the wave of electrical excitation throught the ventricles. Shortly after the QRS complex begins the ventricles start to contract. The third wave is a dome shaped upward deflection called the T wave. It indicates ventricular repolarization and occurs just before the ventricles start to relax. The T wave is smaller and more spread out than the QRS complex because repolarization occurs more slowly than depolarization. Usually repolarization of the atria is not evident in an ECG because it is buried in the larger QRS complex.

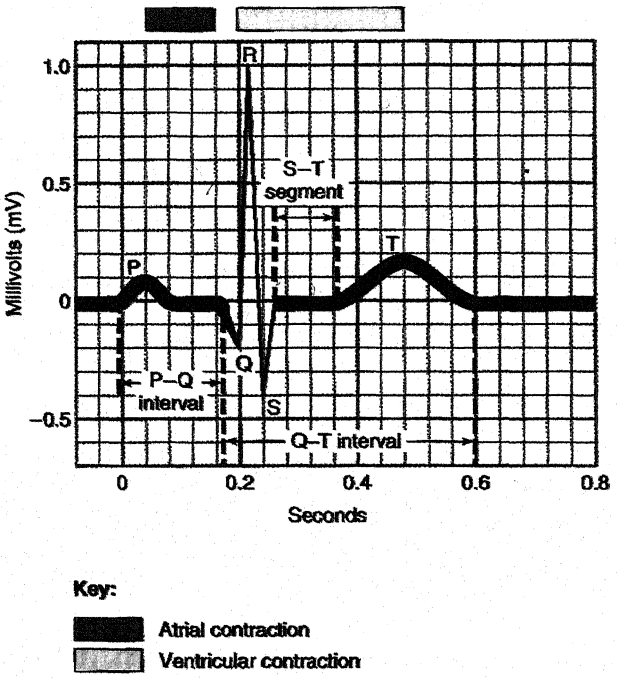


Figure 2.8 : Normal ECG Waveform (Lead II).

In reading an electrocardiogram [31], it is important to note the size and timing of the waves. Larger P waves [32] for example, indicate enlargement of an atrium, as may occur in mitral stenosis. In this condition, the mitral valve narrows, blood backs up into the left atrium, and there is expansion of the atrial wall. An enlarged Q wave may indicate a myocardial infarction (heart attack). An enlarged R wave generally indicates enlarged ventricles. The P-Q (PR) interval is measured from the beginning of the ORS complex. It represents the conduction time from the beginning of atrial excitation to the beginning of ventricular excitation. The P-Q interval is the time required for an impulse to travel through the atria, atrioventricular node and the remaining fibers of the conduction system. In coronary artery disease and rheumatic fever, scar tissue may form in the heart. As the impulse detours around scar tissue, the P-Q interval lengthens.

The S.T segment begins at the end of the S wave and ends at the beginning of the T wave. It represents the time when the ventricular contractile fibers are fully depolarized, during the plateau phase of the impulse. The ST segment is elevated (above the baseline) in acute myocardial infarction and depressed (below the baseline) when the heart muscle receives insufficient oxygen.

The T wave represents [33] ventricular repolarization. It is flatter than normal when the heart muscle is receiving insufficient oxygen, for example, in coronary artery disease. It may be elevated in hyperkalemia (increased blood K^+ level).

Sometimes it is necessary to evaluate the heart's response to the stress of physical exercise. Such a test is called a stress electrocardiogram or stress test. It is based on the principle that narrowed coronary arteries may carry adequate oxygenated blood while a person is at rest, but during exercise will be unable to meet the heart's increased need for oxygen, creating changes that can be noted on an electrocardiogram.

2.4.1 THE NORMAL ECG WAVE

A typical EGG [10, 13, 28] tracing of a normal heartbeat consists of a P wave, a QRS complex and a T wave. A small U wave is not normally visible. Body is a volume conductor i.e. body fluids are good conductors of electricity; therefore, electrical changes occurring in the heart with each heart beat are conducted all over the body and can be picked up from the body surface. The record of the electrical fluctuations during cardiac cycle is called as

Electrocardiogram (ECG)

Thus, the ECG recorded at the surface of the body represents the resultant activity in the individual myocardial fiber.

The waves associated with electrical activity of the heart during each cardiac cycle are represented by letter P, Q, R, S and T.

I. 'P' wave [32] is due to atrial depolarization and precedes atrial systole.

II. 'Q','R' and 'S' waves together constitute the QRS complex and are due to ventricular depolarization. It precedes ventricular systeole.

III. 'T' wave [33] is due to ventricular repolarisation.It coincides with closure of semilunar valves.

2.4.2 ECG RECORDING CONVENTIONS

1. ECG is recorded on mm square graph paper moving at a speed of 25 mm/sec. 'X-axis' represents the time therefore 1mm=0.04 sec (along the X-axis) 'Y-axis' represents the voltage therefore 1mm=0.1Mv (along the Y-axis).

2. Any deflection of the record above the baseline is regarded as positive deflection and any deflection below the baseline is regarded as negative deflection. No deflection from the baseline means the isoelectric line or the isoelectric segment.

3. Spread of the excitation wave i.e. depolarization process towards the electrode gives an upward deflection (positive deflection); and spread of excitation wave away from it causes a downward (negative) deflection.

2.4.3 WAVES ASSOCIATED WITH ECG

The waves associated with the electrical activity of the various parts of the heart tissue during each cardiac cycle are represented by letters P, Q, R, S, T and U.

Axis

The axis is the general direction of the electrical impulse through the heart. It is usually directed to the bottom left, although it can deviate to the right in very tall people and to the left in obesity. Extreme deviation is abnormal and indicates a bundle branch block, ventricular hypertrophy or (if to the right) pulmonary embolism. It also can

diagnose dextrocardia or a reversal of the direction in which the heart faces, but this condition is very rare and often has already been diagnosed by something else(such as a chest x-ray).

'P' wave

I. 1st wave of ECG of duration 0.1 sec; directed upwards, rounded or pointed.

II. It is due to atrial depolarization and represents the spread of impulses from 'SA node' to atrial muscles.

III. Its peak represents invasion of 'AV node' by excitation process.

IV. It occurs just before the 'C' wave of atrial pressure changes during cardiac cycle.

V. Its height is 0.5mV which represents the functional activity of atrial muscles.

VI. If bifurcated or absent, it is regarded as abnormal.

VII. The P wave is the electrical signature of the current that causes atrial contraction. Both the left and right atria contract simultaneously. Irregular or absent P waves may indicate arrhythmia [32]. Its relationship to QRS complexes determines the presence of a heart block.

P-R segment

Following the 'P' wave there is a brief isoelectric period of 0.04sec, called P-R segment.

QRS Complex

I. It is due to the ventricular depolarization.

II. It is completed just before the opening of the semilunar valves.

III. Atrial repolarisation activity merges with the QRS complex.

IV. QRS complex corresponds to the current that causes contraction of the left and right ventricles, which is much more forceful than that of the atria and involves more muscle mass, thus resulting in a greater ECG deflection.

Abnormalities in the QRS complex may indicate bundle branch block [34] (when wide), ventricular origin of tachycardia, ventricular hypertrophy or other ventricular

abnormalities. The complexes are often small in pericarditis.

'Q' wave

I. It is small negative deflection of height less than 0.2mV and duration less than 0.04 sec.

II. Beginning of 'Q' wave represents invasion of mid-portion of the interventricular septum by excitation process.

III. The Q wave, when present, represents the small horizontal (left to right) current) as the action potential travels through the interventricular septum. Very wide and deep Q waves do not have a septal origin, but indicate myocardial infarction.

'R' wave

I. Prominent, positive wave.

II. Its upstroke coincides with the onset of ventricular systole.

III. It represents excitation process suddenly invadig both ventricles i.e. interventricular apex and major portion of both ventricles.

IV Its height is directly proportional to the functional activity of ventricles.

'S' wave

I. Negative deflection which follows the 'R' wave.

II. It represents excitation of more basal part of ventricles.

III. The Rand S waves indicate contraction of the myocardium.

Thus the QRS complex extends from the beginning of 'Q' wave to end of 'S' wave with 0.08 to 0.12 sec duration and height 1.5 to 2mV duration

If its duration is more than 0.12 sec, it indicates heart block i.e. conduction in both or one of the branches of the bundle of His.

S-T segment

Following QRS complex there is a long isoelectric period which extends from the end of 'S' wave to the beginning of 'T' wave called S-T segment. Its duration is 0.04 to 0.08 sec. The ST segment connects the QRS complex and the T wave. It can be depressed in ischemia and elevated in myocardial infarction, and downslopes in digoxin use.

'T' wave

I. Rounded positive deflection of duration 0.27 sec and 0.5 mV height.

II. It represents ventricular repolarisation.

III. End of T-wave coincides with the closure of the semilunar valves.

IV. In most leads, the T wave is positive. Negative T waves can be signs of disease, although an inverted T wave is normal in V1 (and V2-3 in black people).

V. T wave abnormalities [35, 36, 37, 38, 39] may indicate electrolyte disturbance, such as hyperkalemia.

Iso-electric period

Following T-wave is a brief isoelectric period of 0.04 sec.

'U' wave

I. Rarely seen, as positive small [40] round wave of 0.08sec duration and 0.2 mV height.

II. It is due to slow repolar4isation of papillary muscles.

PR interval

I. Interval from the beginning of 'P' wave to the beginning of Q or R wave (if Q is absent).

II. It represents atrial depolarization plus conduction time of bundle of His.

III. Normal duration 0.13 to 0.16 sec at a heart rate (HR) of 72/min, duration decreases with increase in HR.

IV. If duration is more than 0.2 sec indicates delayed conduction in bundle of His.

V. Duration of less than 0.13 sec indicates impulse has probably arisen in the AVN.

QT interval

I. Interval [41, 42, 43]from the beginning of 'q' wave to the end of T-wave, normal duration [44] of 0.40 to 0.43 sec.

II. It represents ventricular depolarization and repolarisation.

ST interval

I. (QT-QRS comlex) i.e. end of 'S' wave to end of 'T' wave, normal duration 0.32 sec.

II. It represents ventricular repolarisation.

TP segment

I. Period from the end of 'T' wave to the beginning of 'P' wave of next cardiac cycles.

II. It represents polarized state of whole heart.

III. Its duration is inversely related to H.R. Normal is 0.2sec @ H.R 75/min.

J point

I. Point between 'S' wave and ST segment.

II. It is point of 'no' electrical activity.

2.5 ARRHYTHMIA

Arrhythmia (a-RITH-me-a) is a general term that refers to an abnormality or irregularity in the heart rhythm. Some physicians use the term dysrhythmia since that implies an abnormal rhythm, whereas arrhythmia implies no rhythm. An arrhythmia results when there is a disturbance in the conduction system of the heart. It may be due to either faulty production of electrical impulses as they pass through the system.

2.5.1 MECHANISMS THAT CAUSE ABNORMAL IMPULSES

Automaticity

Automaticity refers to a cardiac muscle cell firing off an impulse on its own. Every cardiac cell has this potential: if it does not receive any impulses from elsewhere, its internal pacemaker" will fire off an impulse after a certain amount of time. A single specialized location in the atria, the sinoatrial node, has a higher automaticity (a faster pacemaker) than the rest of the heart, and therefore is usually the one to start the heartbeat.

Any part of the heart that initiates an impulse without waiting for the sinoatrial node is called an ectopic focus, and is by definition a pathological phenomenon. This may cause a single premature beat now and then, or, if the ectopic focus fires more often than the sinoatrial node, it can produce a sustained abnormal rhythm. Rhythms produced by an ectopic focus in the atria [45], or by the atrioventricular node, are the least dangerous [46] arrhythmias; but they can still produce a decrease in the heart's pumping efficiency [47], because the signal reaches the various parts of the heart muscle with slightly different timing than usual and causes a poorly coordinated contraction. Conditions that increase automaticity include sympathetic nervous system stimulation and hypoxia. The resulting heart rhythm depends on where the first signal begins: if it is the sinoatrial node, the rhythm remains normal but rapid; if it is an ectopic focus, many types of arrhythmia [48] can result.

Reentry

Reentrant arrhythmias occur when an electrical impulse travels in a circle within the heart, rather than moving outward and then stopping. Every cardiac cell is able to transmit impulses in every direction, but will only do so once within a short period of time. Normally the impulse spreads through the heart quickly enough that each cell will only respond once, but if conduction is abnormally slow in some areas, part of the impulse will arrive late and will be treated as a new impulse, which can then spread backward. Depending on the timing, this can produce a sustained abnormal rhythm [49], such as atrial flutter, a self-limiting burst of supraventricular tachycardia, or the dangerous ventricular tachycardia [50].

2.5.2. CAUSES OF ARRHYTHMIAS

* Factors [51] such as caffeine, nicotine, alcohol, anxiety, certain drugs, hyperthyroidism, potassium deficiency and certain heart diseases [52].
* When the heart's natural pacemaker develops an abnormal rate or rhythm.
* When the normal conduction pathway is interrupted.
* When another part of the heart takes over as pacemaker.

* Coronary artery disease, high blood pressure, diabetes, smoking, excessive use of alcohol, drug abuse and stress.

* Certain substances, including prescription medications, dietary supplements and herbal remedies are known to cause arrhythmias in some people.

2.5.3 CLASSIFICATION OF ARRHYTHMIAS

Abnormal rhythms [1] occur as primary and secondary disorders. Primary disorders of rhythm reflect a basic, essential abnormality. Secondary disorders of rhythm only occur as a result of, and secondary to, a primary disorder.

The primary disorders of rhythm (33) may, in simplified form, be classified into two major categories :

1. Disturbances of impulse formation
2. Disturbances of impulse conduction.

Disturbances of impulse formation generate following arrhythmias :

Sinus Rhythms

Sinus arrhythmia

Sinus tachycardia

Sinus bradycardia

Ectopic atrial rhythms

Atrial Extrasystoles

Paroxysmal atrial tachycardia

Atrial Fibrillation

Atrial flutter

AV nodal rhythms

AV nodal extrasystoles

Extrasystolic-paroxysmal- AV nodal tachycardia

Idionodal tachycardia

Ventricular rhythms

Ventricular Extrasystoles

Extrasystolic ventricular tachycardia

Idioventricular Tachycardia

Ventricular flutter

Ventricular fibrillation

Ventricular parasystole

Disturbances of impulse conduction generates following arrhythmias

SA block

AV block

The Wolff- Parkinson white syndrome

Reciprocal rhythms

The secondary disorders of rhythm

Escape Rhythms

Atrial Escape

AV nodal Escape

Ventricular Escape

AV dissociation

Phasic aberrant ventricular conduction

2.5.4 FUNDAMENTAL DESCRIPTIVE PROPERTIES OF CARDIAC RHYTHMS

1 the rhythm [1] has a anatomical origin. The impulse may rise in the SA node, the atria, the AV node and ventricles.

2 The rhythm has a discharge sequence: normal inherent discharge, (as would occur normal sinus rhythm or an idioventricular escape rhythm), tachycardia, bradicardia extrasystole, parasystole, flutter or fibrillation.

3 The rhythm has a conduction sequence: for example, 2:1 AV block, complete AV block, 2:1 SA block.

DUAL RHYTHMS

A dual rhythm is a rhythm wherein two pacemakers concomitantly contribute to the rhythm of the heart. A dual rhythm is present in every form of AV dissociation – one pacemaker activating the atria and the other the ventricles. When this occurs, the descriptive properties of both the dissociated rhythms must be stated.

Example

1. Sinus rhythm with complete AV block and an idioventricular escape rhythm.

2. Ventricular tachycardia with AV nodal interference dissociation from normal sinus rhythm.

ANALYSIS OF CARDIAC RHYTHMS

On the basis of the aforementioned principles cardiac rhythm [53, 54] nay be fundamentally analysed as follows:

1. The atrial deflexion is defined and analysed to determine whether it represents;
   (a) a normal P wave,
   (b) an ectopic or P deflexion,
   (c) a flutter –F- wave,
   (d) a chaotic fibrillation –f- wave.
2. The atrial rate is determined.
3. The regularity of the atrial rhythm is determined.
4. The relationship of the atrial deflexions to the QRS complexes is the determined.
5. The QRS configuration is analysed.

THE GRAPHIC REPRESENTATION OF THE INTRACARDIAC CONDUCTION

Intracardiac conduction [1] may be conveniently and conventionally represented by means of a 'ladder' diagram. Refer figure 2.9. This is a graphic representation:

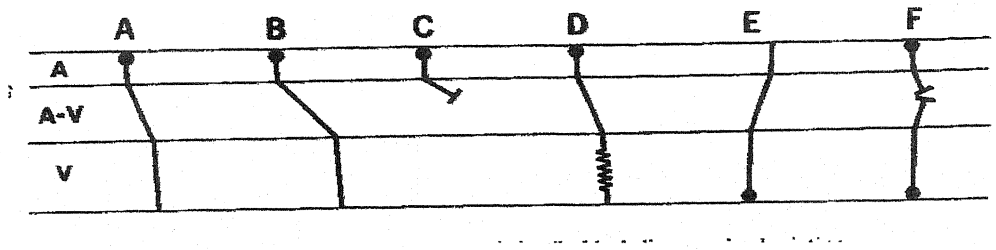


Figure 2.9 : Graphic representation of the use of the 'ladder diagram in depicting arrhythmias'.

The ordinate represents the anatomic levels of SA node, atria, AV node and ventricles; the abscissa represents time.

A illustrates normal conduction of a sinus impulse – black dot – arise in the SA node and is conducted relatively quickly through the atria (A), as reflected by the relatively steep slope. The impulse delayed within the AV node or junction (A-V), as reflected by the gradual slope, and is finally conducted relatively steep slope.

First degree AV block – prolonged P-R interval –a delay in conduction through the AV junction. This reflected by an even shallower slope.

B, Represents AV block – an interruption of conduction within the AV node.

A, B and C together would represent sinus rhythm complicated by 3:2 second degree AV block of the wenckebach type.

D, Represents phasic aberrant ventricular conduction –the abnormal intermittent intraventricular conduction of a supraventricular impulse.

E represents a ventricular impulse with retrograde AV conduction to the atria.

F represents a ventricular impulse, which is dissociated from a near-**synchronous** sinus impulse. Interference with consequent AV dissociation occurs within the AV node.

2.5.8 SOME IMPORTANT ARRHYTHMIAS

The major life threatening arrhythmias are explain as follows

1 Normal Sinus Rhythm

Normal sinus rhythm is reflected by the inscription of normal P waves at a rate [55] which ranges between 60 and 100 per minute. Normal sinus rhythm is usually associated with normal intraventricular conduction, and is thus reflected by the sequential inscription of P-QRS-T complexes.

2 Sinus Arrhythmia

Sinus arrhythmia is characterized by alternating periods of slow and rapid rates; [1, 55] it is due to an irregular fluctuating discharge of the SA node. The condition is most commonly associated with the phases of respiration – respiratory sinus arrhythmia. The period of faster rate occur towards the end of inspiration and the periods of slower rate towards the end of expiration. The mechanism is meditated by reflex stimulation of the vagus nerve from receptors in the lungs.

Diagnosis

The impulse rise from the SA node and the P waves are therefore normal; the subsequent course of the sinus impulse, on the absence of a conduction disturbance, is also normal, resulting in a normal P-R interval and QRS-T complex. The arrhythmias is thus characterized by normal P-QRS-T complexes, with alternating periods of gradually lengthening and gradually shortening P-P intervals [1, 55].

Sinus arrhythmia is accentuated by vagotonic procedures, such as digitalis administration and carotid sinus compression. It is abolished by vagolytic procedures, namely exercise, atropine and amyl nitrate.

Significance

Respiratory sinus arrhythmia [56] is a normal physiological; phenomena and is most marked in young persons. It may cause considerable irregularity of the pulse in childhood [57].

3 Sinus tachycardia

Sinus tachycardia [58] occurs when the SA node discharges at a rate faster than 100 per minute in the adult. The normal resting rate in infant's averages 120-130 beats per minute. Slowing gradually to reach the adult rate at puberty.

Diagnosis

Sinus tachycardia is, in the absence of a complicating conduction disturbance, characterized by normal P-QRS-T complexes, which are recorded in rapid succession. It varies with emotion, respiration and exercise. Vagotonic procedures, e.g. carotid sinus compression, result in slight but gradual slowing.

Significance

Sinus tachycardia [59] is a normal physiological response to exercise and emotion. A sinus tachycardia that persists at rest usually and expression of some underlying disorder. It occurs in anxiety states, thyrotoxicosis, toxaemia, cardiac failure (as a result of an increased Bainbridge reflex) and acute carditis. It is a normal accompaniment of fever. The sinus rate will increase by 8 beats per minute for every one-degree increase in temperature. A diminution in oxygen saturation, as occurs at high altitudes or in association with congenital heart disease, will also cause a sinus tachycardia. Failure to develop sinus tachycardia with exercise or fever may be an expression of structural nodal disease – the so-called sick sinus syndrome. It may be caused by the administration of adrenaline, atropine, caffeine and amyl nitrate.

4 Sinus bradycardiya

Sinus bradycardia, occurs when the SA node discahges at a rate slower than 60 per minute.

Diagnosis

Sinus bradycardia [60], in the absense of a complicating conduction distribution, is charactersised by normal P-QRS-T complexes, which are recorded in slow succession. It is commonly associated with respiratory sinus arrhythmias.

Significance

Sinus bradicardia occurs as normal phenomena in athletes. Slowing of the sinus rate – at times to bradycardia levels – is the physiological response to sleep. Sinus bradycardia is accentuated by digitalis and Vagotonic procedures, such as carotid sinus compression. The rate quickens gradually with exercise, emotions and amyl nitrate.

Sinus bradycardia is associated with myxoedima [1], obstructive jaundice (the effect of direct action of the bile salts on the SA node) uraemia, (increased and persistence oculocardiac reflexes)

A common present-day cause of sinus bradycardia is the administration of beta-blocking agents [1].

5 Atrial extrasystoles

An atrial extrasystole [2] is due to the premature discharge of an ectopic atrial focus. It has the following characteristics.

Bizarre P wave

The discharge arises from an ectopic atrial focus, i.e from a point other than the SA node. The activation front thus travels across the atria by unusual pathways, resulting in an abnormal or bizarre P' wave – a P' wave that is differt from the sinus P wave and which may be pointed, notched, biphasic or inverted.

The ectopic impulse arises prematurely, i.e in the diastolic period of the preceding sinus beat, and is thus recorded earlier than the next anticipated sinus P wave.

6 Heart Block

One serious arrhythmia [1] is called a heart block [61, 62]. Perhaps the most common blockage is in the atrioventricular (AV) node, which is the only path for impulses in the atria to reach the ventricles. This disturbance is called atrioventricular (AV) block. In first-degree AV block, P-Q (PR) interval [63]is prolonged, usually because conduction through the AV node is slower than normal. In second-degree AV block, some of the SA node impulses are not conducted through the AV node. This results in "dropped" beats, since excitation doesn't reach the ventricles. In third-degree (complete) AV block, none of the SA node impulses get through the AV node. Autorhythmic cells in the atria and ventricles pace the upper and lower chambers independently. With complete AV block [21, 22], the ventricular contraction rate is less than 40 beats/min. Due to decreased cardiac output and diminished brain blood flow, patients my experience dizziness, unconsciousness or convulsions.

7 Ventricular Arrhythmias

When pacemakers in the SA node and in the AV junction both fail then the ventricular pacemakers, which have the lowest intrinsic rate, are able to take over. This usually occurs at a rate of 20-40 beats per minute, These pacemakers may also be manifest on occasions when the normal beat fails to reach the ventricles (e.g., in complete heart blocks); the result may be a ventricular escape [23] beat or a series of beats at the escape rhythm.

Premature ventricular complex (PVC) beats are very common in both health and disease. Activation tends to spread outward from the Ventricular [64, 65]ectopic site of activation mainly by cell-to-cell conduction. Consequently, the QRS duration is abnormally long (greater than 0.12s) and the QRS contour is abnormal [66]. There may also be lack of a discernible P wave preceding the QRS. Following the PVC is a compensatory pause. This simply refer to the fact that the basic sinus rate is unaffected by the PVC; consequently since the PVC prevents one sinus beat from reaching the ventricle when the next arrives a greater than PR interval will have elapsed.

8 Ventricular tachycardia

Ventricular tachycardia is a very serious arrhythmia [58, 67] arising most frequently in association with the occlusion of a coronary artery and/or electrolyte disturbance. It usually lies in the range 130 to 180 beats per minute, This arrhythmia arises from an enhanced automaticity of focal ventricular tissue or by reentry, which involves slow conduction and/or block that enables activation to return to an normal site beyond the refractory period permitting subsequent loop circuits to be made.

9 Ventricular Fibrillation and flutter

Definition

Ventricular fibrillation [65, 68] is a disorganized and chaotic activity of the heart, which results in irregular and deformed deflections of varying height, width and shape. This condition is terminal unless cardioverted. Ventricular flutter is a very rapid and regular ectopic ventricular discharge with an abnormal intraventricular conduction resulting in a wide, bizarre and sine like QRS complex fused with The T wave.

Causes

1. Coronary heart disease.
2. Drugs : Digitalis, adrenaline and anesthetics (79).
3. During cardiac surgery, due to hypoxia.
4. Hypothermia
5. Electric Shock

Diagnosis

Bizarre ventricular pattern of different shapes and sizes.

10 Ventricular Premature Contraction (VPC)

Another form of arrhythmia arises when a small region of the heart outside the pacemaker (an ectopic focus) becomes more excitable than normal, causing an occasional abnormal impulse to rise between normal impulses. As a wave of depolarization spreads outward from the ectopic focus, it causes a ventricular premature contraction (VPC). The contraction occurs early in diastole before the SA node is normally scheduled to discharge its impulse. VPCs ay be relatively benign and may be caused by emotional stress, excessive intake of stimulants such as caffeine or nicotine and lack of speed. In other cases, the contractions may indicate an underlying pathology.

11 Supraventricular Premature Beats Or Extrasystoles (SVPB)

Definition

SVPB [21, 22] occurs due to a premature discharge of an ectopic focus, situated above the ventricles, either in the atrium or the AV node.

Characteristics

1. An atrial beat occurs prematurely so that a P wave is recorded earler than the anticipated P wave.
2. Premature atrial excitation lewads to an alternation in the P wave and the PR interval as the impulse travels along unusual pathways. The P wave may be upright, inverted or diphasic.

3. The premature beat usually initiates a ventricular complex which resembles the normal beat; hence the QRST complex of the premature beat resembles the QRST complex of the normal beat.

4. The compensatory pause is incomplete i.e. the sum total of the R-R intervals of the normal beat preceding and following a premature beat is not double the normal R-R interval.

Causes

1. Idiopathic
2. Heart diseases: coronary, rheumatic, thyrotoxicosis, diphtheria and hypertension.
3. Excessive use of tea, coffee, tobacco and alcohol.
4. Drugs: digitalis, amphetamine, adrenaline, thyroxine and emetine [49].
5. Anoxaemia: anaemia, shock.
6. Reflex: peptic ulcer, kidney and gall bladder stones.
7. Manipulation of intrathoracic organs during surgical procedures on the heart or thoracic organs.

12 PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (SVT)

Paroxysmal supraventricular tachycardia [1] is characterized by normal QRS but inverted P waves in leads II, III, and aVp along with a heart rate most frequently in the range 170-220 beats per minute. The most common mechanism involves patients with dual AV nodal pathways in which one has fast conduction and long refractory time while the other has slow conduction and rapid recovery. A premature atrial impulse will result in blocked conduction on the fast pathway and consequently a lengthened PR interval due to conduction on the, slow pathway. The impulse may then enter the fast pathway, conducting retrograde, and ultimately reexcite the slow link to establish a closed reentry loop.

Under unusual circumstances the normal rate may become so slow that a site in the AV junction may become an active source of a cardiac beat. This phenomenon is described as junctional escape.

Junctional escape is easy to recognize from its physiological properties. These include normal QRS complexes, a long pause preceding the escape arising from the

underlying bradycardia and retrograde activation of the atria resulting in an inverted P wave.

A situation sometimes arises when the atria are depolarized by impulse initiation from the SA node in the normal way but the ventricles are depolarized from a junctional pacemaker at a somewhat higher rate, Under these conditions retrograde conduction in the AV tissue is inhibited because of the preceding sinus impulses. The result is that the atria and ventricles beat completely independently.

Definition

SVT is a series of three or more SVPBs which may occur for a few beats or continuously for several hours or days. The last beat of the series is followed by a compensatory pause that is incomplete. Usually the rhythm is regular and at a rate of 160-220/min.

Mechanism

The exact mechanism of SVT is not known but two theories have been proposed:

1. Ectopic Mechanism: An ectopic focus, in either of the atria discharges regularly at the rate of 160-220/min. At this rate each atrial stimulus activated the ventricular muscle resulting in a regular ventricular rhythm.

2. Re-entry Mechanism: The tachycardia is initiated by an extra systole with a prolonged PR interval but there is normal conduction in the AV node and the ventricles. The stimulus reenters the bundle of his in a retrograde direction and stimulated non-refractory sited in the bundle. It is then propagated in a normal anterograde direction which results in a second ventricular capture. Perpetuation of the stimulus along this pathway causes SVT.

Diagnosis

SVT (60) is a continuous run of SVPBs so that each P wave is followed by a QRS complex.

The spread of the impulse through the atrial muscles occurs more slowly than the normal sinus beat or SVPB. Hence the PR interval is prolonged and the P wave may be obscured by the preceding QRS complex simulating junctional tachycardia.

Causes

Same as SVPB.

Significance

SVT may last for a few seconds to several days. It is usually benign and if without an underlying cause does not reduce life expectancy. Persistence of SVT in a patient with organic heart disease may lead to cardiac failure and coronary insufficiency. Persistance for a very long period, even in a normal individual, may cause cardiac failure.

13 Ventricular Premature beat or Extrasystole (VPB)

Definition

VPB occurs due to premature discharge [21, 22] of an ectopic focus in the ventricles.

Characteristics

1. The beat arises prematurely.
2. since the impulse originates in the ventricles and does not activate the atria, the P wave is absent.
3. The QRS complex is wide, bizarre and tall, with t waves in the opposite direction as the major deflection of the QRS complex i.e. if the R wave is prominent, the T wave is inverted and if the S wave is prominent, the T wave is upright.
4. The compensatory pause is complete because VPB does not depolarize the SA node. The impulse from the SA node following a VPB does not activate the ventricles as they will be in the refractory period. The ventricles will respond only to the next sinus impulse and hence the interval between the two sinus beats preceding and following the VPB will be exactly twice the normal interval between two sinus beats.

Types

1. Single isolated extrasystole [21].

2. Bigeminy or coupling : The extrasystole occurs regularly after every sinus beat [71.72,73,74,75]. If it occurs every second sinus beat it is called trigeminy.

3. In salvos : Two or more extrasystoles [21, 22] occur in succession. This may lead to paraxymal tachycardia.

4. Interpolated : The extrasystole occurs in between two normal beats without any compensatory pause.

5. Multifocal : The extrasystoles arise from different foci, so that the morphology of the extrasystole varies in the same lead.

6. Reciprocal beat : The extrasystole spreads from the AV node to the ventricles and upwards into the atria. When it reaches the AV node it reverses its direction and reenters the ventricles causing a second ventricular contraction. Thus two QRS complexes separated by a nodal P waves are recorded.

7. Parasystole : The impulse simultaneously arises from the normal pacemaker and the ectopic focus. The two rhythms occur regularly but independent of each other so that the coupling distance between the ectopic focus and the previous sinus beat varies. At times, a fusion or summation beat occurs, the morphology of which is a combination of the sinus and the ectopic beat.

8. Blocked atrial premature beat : The P wave occurs during the refractory period of the AV node and the ventricles and hence these beats are blocked. However, the early P wave alters the morphology of the preceding T waves.

Significance

An extrasystole can occur in a normal person without any lesion of the heart. Usually it is benign and of no significance. However it is significant if:

1. It occurs for the first time after the age of 40
2. It is associated with a heart lesion.
3. It is multifocal.
4. It occurs more than 5 times per minute.
5. There is R on T phenomenon.
6. It occurs in salvos of 2 or more.
7. It occurs following exercise.

Causes

Same as VPB.

14 Paroxysmal Ventricular Tachycardia (VT)

Definition

VT is a series of three or more VPBs which may occur for a few beats or continuously for several hours or days. The last beat of the series is followed by a compensatory pause that is complete. Usually the rhythm is regular at 160-220 /min.

Diagnosis

VT [50] is a continuous run of VPBs with QRS complexes smoothly merging with the ST segment and T waves giving an appearance of large wide undulations which are irregular.

Causes

Same as SVBP.

15 Atrial Flutter And Fibrillation

Definition

Atrial flutter [76] is a rapid and regular contraction of the heart at a rate of about 220-350/min. Varying degrees of AV block lead to a much slower ventricular rate. The P waves of the atrial flutter have a saw-tooth appearance and are called flutter waves.

Atrial fibrillation [77] is a chaotic rhythm of the atria which causes small twitches of the atrial myocardium instead of an active atrial contraction which normally aids ventricular filling phase of the ventricular diastole.

Mechanism

AV conduction of the atrial impulse depends upon the atrial rate. With atrial rates 160-220/min, (atrial tachycardia), vagal stimulation leads to asystole followed by reversion to sinus rhythm. With rates 220-350/min. (atrial flutter), varying degrees of AV block occur without a change in atrial rate [76]. With rates above 350/min. (atrial fibrillation), the atrium cannot respond completely too each stimulus and a chaotic disturbance occurs. Majority of the atrial impulses reaching the AV node are blocked. An

occasional impulse occurs during the non-refractory period and is conducted to the ventricles leading to an irregular ventricular rhythm.

With atrial rate less than 200/min.P waves of the atrial flutter resemble those of SVT. As the atrial rate increases, a more prominent T wave in a direction opposite to the P wave appears. With increasing atrial rates the T wave amplitude increases and at rate 300/min. It will be equal in amplitude to the P wave resulting in a saw-tooth appearance.

Causes

Comman	**Uncommon**
1. Rheumatic fever	1. Constrictive pericarditis
2. Coronary heart disease	2. Cor-pulmonale
3. Thyrotoxicosis	3. Bronchogenic carcinoma
4. Diptheria	4. A.S.D.
5. Drugs: digitalis, adrenaline, emetine [78]	5. Hypertension
6. Excessive use of tea, coffee, tobacco and alcohol .	6. Lone atrial fibrillation. 7. W.P.W. Syndrome.

Diagnosis

Atrial Flutter

1. Fast atrial rate of 220-350 / min. with ventricular rate half or one forth of the atrial rate
2. P waves replaced by flutter waves.
3. Ventricular rhythm usually regular, unless there is a changing AV block.

Atrial Fibrillation

1. Irregularly irregular ventricular rhythm [79].
2. P waves replaced by fibriilation waves.
3. Normal QRS complexes.

At times the rhythm may alternate between flutter and fibrillation and a precise difference cannot be discerned. This is called 'flutter fibrillation'.

Chaotic Atrial Rhythm :

When there are more than three morphologically different P waves [80, 81] that activate the ventricles, it is called chaotic atrial rhythm. It resembles wondering pacemaker but unlike the latter there is no dominant P wave. This rhythm [82, 83] is seen in elderly persons with chronic obstructive pulmonary disease or coronary artery disease.

2.5.6. SUMMARY OF COMMONLY OCCURRING LIFE THREATENING ARRHYTHMIAS

Refer figure 2.10 for Arrhythmia Waveforms (Lead II)

The commonly occurring life threatening arrhythmias are explained below in brief:

Asystole

It is a condition in which there is lack of conduction for an extended duration.

Fusion beat

It is a parasystolic condition in which two pacemakers in heart (Say SA and AV) discharge at their own inherent rate, occasionally causing simultaneous invasion of ventricular musculature, each activating part of ventricles. The resulting QRS complex has a configuration intermediate below 'pure' sinus beat and 'pure' ventricular beat. The resulting summation complex is known as fusion beat [84, 85].

Missed beat

In second degree AV block, transmission through the conducting system becomes difficult until it falls completely and a beat is 'dropped'. The condition is noted if R-R interval is greater than 2 R-R

Atrial fibrillation

In this excitation and recovery of atria are disorganized and chaotic.

Ventricular fibrillation

Unconditional, chaotic, uncoordinated fluttering of ventricles [20, 86] is called ventricular fibrillation. No defined P-QRS-T signal is observed.

Bigeminy

Presence of ventricular premature beat [87, 88, 89, 90] between alternate normal beat. The VPB occurs very early i.e. AV node still partially refractory. Following sinus beat occurs on time but relatively late in relationship to the extrasystole.

Multifocal ventricular extrasystoles

Extrasystole that arise from different foci and consequently give rise to different QRS complexes are termed as multifocal ventricular extrasystoles. Usually these are indication of serious myocardial disease.

R on T wave

Ventricular extrasystoles may rarely occur with very short coupling interval and will consequently coincide with and be superimposed upon or near the apex or the distal limb of preceding T wave. These are more prone to ventricular fibrillation [91]occurring in context of acute myocardial infarction.

Ventricular Tachycardia

It is due to rapid discharge of an ectopic ventricular pacemaking focus. It may be defined as series of three or more consecutive ventricular ectopic beats.

Heart Block

It is a condition in which the conduction of the pacing signal is either delayed or completely blocked resulting in inability to activate the chamber or part being paced. This leads to absence of the characteristic waveform of the chamber i.e. if there is complete AV block, R wave is missing.

VPB1

In this condition there is premature ventricular contraction arising in diastolic period of the preceding sinus beat followed by compensatory pause.

VPB2

Ventricular extrasystole arising from an ectopic focus followed by incomplete compensatory pause.

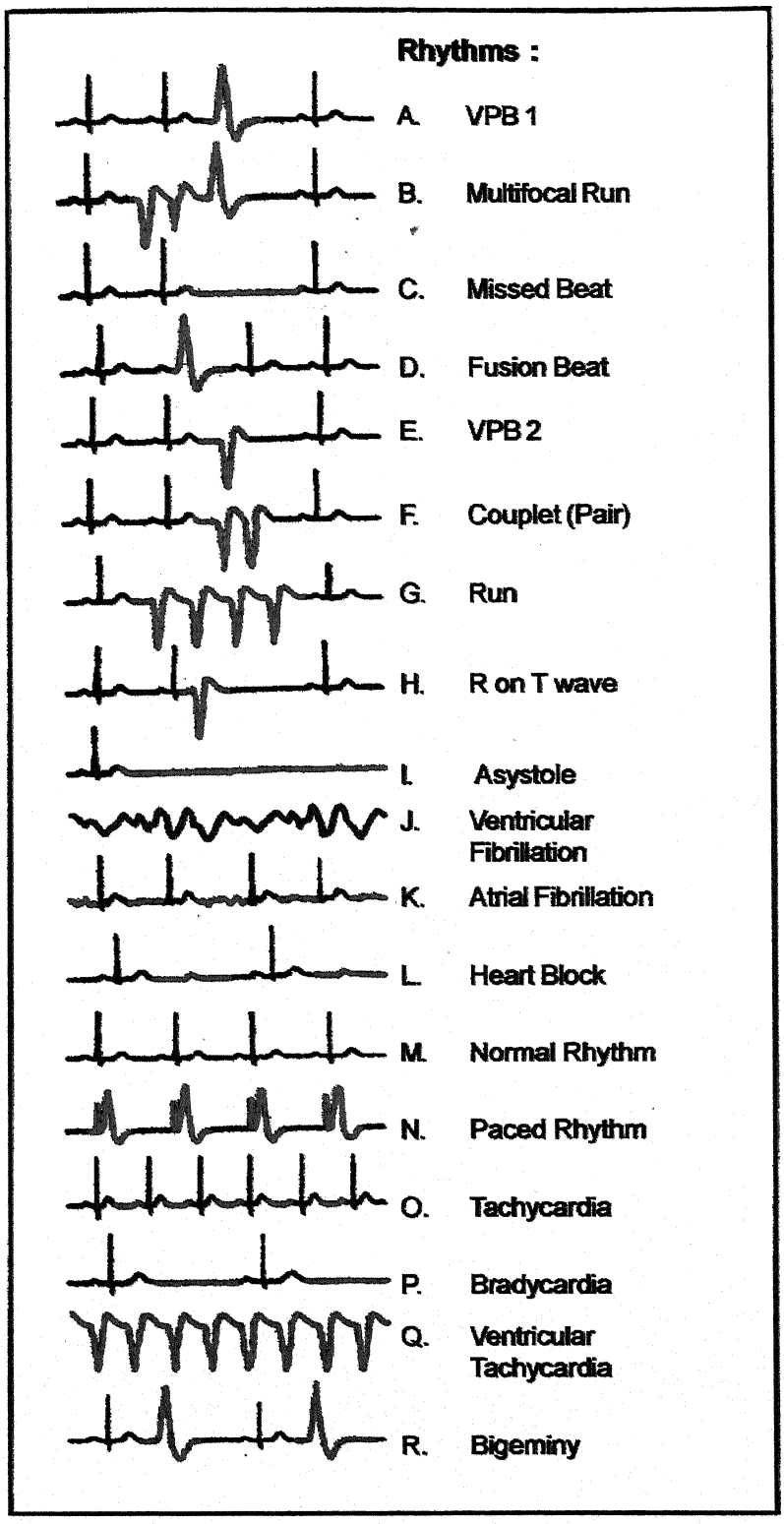


Figure 2.10 : Arrhythmia Waveforms (Lead II)

2.6 CONCLUSION

A survey of various life threatening arrhythmias is done and taking the views of many cardiologists and cardiac surgeons, out of all, seventeen arrhythmias and a normal ECG waveform his selected for simulation. The literature provided in this chapter shows that there is a need of a various simulation programs for arrhythmias as the ECG signal shows changes with the age, health, drug intake by patient, and patient suffering from respiratory diseases.

CHAPTER - III

SIMULATION USING MATHEMATICAL MODELLING OF THE HEART AND LEADS THEORY

3.1. INTRODUCTION

Electrocardiographic (ECG) signals may be measured at any point on the body surface. On the torso the signal magnitude in normal healthy adults is around 5 mV and is, consequently, relatively easy to measure. One of the reasons for the extensive use of the electrocardiogram is, in fact, both its ease of measurement and its noninvasive character.

The morphology of the electrocardiographic signals depends, basically, on the cardiac generators the volume conducting medium and the location of the pickup electrodes at the surface [92] of the body. The latter are referred to as the leads [93]. The location of the leads may be established to fulfill some theoretical consideration (as in the case of orthogonal lead systems) or simply to be defined by anatomical landmarks so that the effect of geometry may enter in a well defined and consistent manner (standard lead system). This chapter describes the ECG leads and simulation of lead II signal of all seventeen arrhythmias using mathematical modeling of the heat and leads theory.

3.2 ECG LEADS

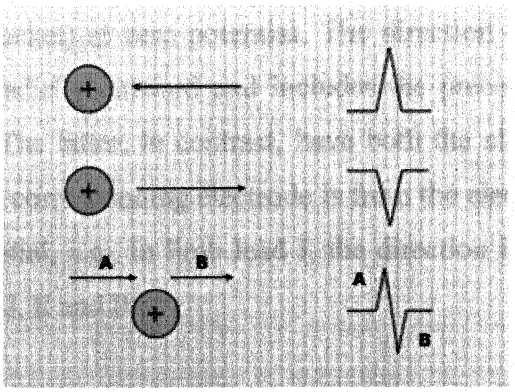


Fig. 3.1 : Graphic showing the relationship between positive electrodes, depolarization wavefronts (or mean electrical vectors), and complexes displayed on the ECG.

An ECG is obtained by measuring electrical potential between various points of the body using a Biomedical Instrumentation amplifier. A lead records the electrical signals of the heart from a particular combination of recording electrodes which are placed at specific points on the patient's body.

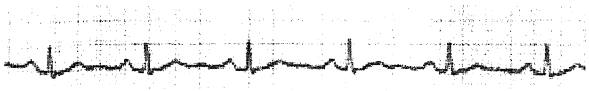
Refer Figure 3.1 for Graphic showing the relationship between positive electrodes, depolarization wavefronts (or mean electrical vectors), and complexes displayed on the ECG

- When a depolarization wavefront (or mean electrical vector) moves toward a positive electrode, it creates a positive deflection on the ECG in the corresponding lead.
- When a depolarization wavefront (or mean electrical vector) moves away from a positive electrode, it creates a negative deflection on the ECG in the corresponding lead.
- When a depolarization wavefront (or mean electrical vector) moves perpendicular to a positive electrode, it creates an equiphasic (or isoelectric) complex on the ECG. It will be positive as the depolarization wavefront (or mean electrical vector) approaches (A), and then become negative as it passes by (B).

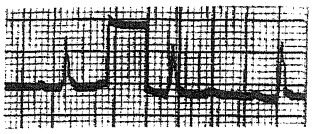
There are two types of leads—unipolar and bipolar [94]. The former have an indifferent electrode at the center of the Einthoven's triangle (which can be likened to a 'neutral' of the wall socket) at zero potential. The direction of these leads is from the "center" of the heart radially outward and includes the precordial (chest) and the limb leads—V_L, V_R & V_F. The latter, in contrast, have both the electrodes at some potential and the direction of the corresponding electrode is from the electrode at lower potential to the one at higher potential, e.g., in limb lead I, the direction is from left to right. These include the limb leads--I, II and III.

3.2.1 Limb leads

Lead I



Lead II



Lead III

Fig. 3.2 : Limb Leads

Leads I, II and III are the so-called **limb leads** (Refer Figure 3.2 for Limb Leads) because at one time, the subjects of electrocardiography had to literally place their arms and legs in buckets of salt water in order to obtain signals for Einthoven'sstring galvanometer. They form the basis of what is known as **Einthoven's triangle.** Eventually, electrodes were invented that could be placed directly on the patient's skin. Even though the buckets of salt water are no longer necessary, the electrodes are still placed on the patient's arms and legs to approximate the signals obtained with the buckets of salt water. They remain the first three leads of the modern 12 lead ECG [69].

- Lead I is a dipole with the negative (white) electrode on the right arm and the positive (black) electrode on the left arm.
- Lead II is a dipole with the negative (white) electrode on the right arm and the positive (red) electrode on the left leg.
- Lead III is a dipole with the negative electrode (black) on the left arm and the positive (red) electrode on the left leg.

Refer Figure 3.3 for Proper Placement of the Limb Leads

If the heart is considered as generating current that flow though closed paths that both leave and return to the heart, then it becomes clear that little current could be expected to both enters and leave the arms and legs. Consequently, to a good approximation, these appendages will be equitpotential and it should make little difference if the limb leads are applied at the wrists and ankles (where they are connected conventionally) or the upper arms and upper thigh. The basic volume conductor for the

heart can be approximated as the thorax and the locations of the standard leads identified with the arms and thigh.

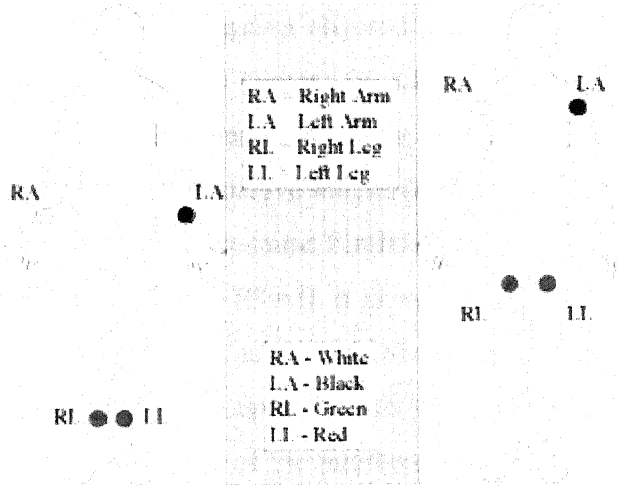


Fig. 3.3 : Proper Placement of the Limb Leads.

Measurement of electrocardiographic voltage requires a lead repair, and corresponding to the left arm (LA), right arm (RA), and left leg (LL) three pairs can be identified. These are defined to be leads I, II, and III, as follows:

Lead I $$V_I = V_{LA} - V_{RA} \quad (21)$$

Lead II $$V_{II} = V_{LL} - V_{RA} \quad (22)$$

Lead II $$V_{III} = V_{LL} - V_{LA} \quad (23)$$

Application of Kirchhoff's loop equations requires that

$$(V_{RA}-V_{LA}) + (V_{LA}-V_{LL}) + (V_{LL}-V_{RA})=0 \quad (24)$$

Substituting the definitions given in Eqs. 21, 22, 23 into the condition stated in Eq. 24 results in

$$V_{III} + V_I = V_{II} \quad (25)$$

Consequently, one of the standard lead signals [13] is redundant. Despite this redundancy, all three are recorded clinically, and this provides an internal check.

3.2.2 Augmented limb leads

Leads aV_R, aV_L, and aV_F are augmented limb leads. They are derived from the same three electrodes as leads I, II, and III. However, they view the heart from different angles (or vectors) because the negative electrode for these leads is a modification of Wilson's central terminal, which is derived by adding leads I, II, and III together and plugging them into the negative terminal of the EKG machine. This zeroes out the negative electrode and allows the positive electrode to become the "exploring electrode" or a unipolar lead. This is possible because Einthoven's Law states that I + (-II) + III = 0. The equation can also be written I + III = II. It is written this way (instead of I + II + III = 0) because Einthoven reversed the polarity of lead II in Einthoven's triangle [13, 69], possibly because he liked to view upright QRS complexes. Wilson's central terminal paved the way for the development of the augmented limb leads aV_R, aV_L, aV_F and the precordial leads V1, V2, V3, V4, V5, and V6

The limb leads can also be measured relative to the central terminal, giving three additional leads. If, say, this is done with the right arm, then eliminating the right-arm resistor from the central terminal can be shown to result in the same signal, but 50% larger. Such a lead is designated aV_R (augmented right arm signal relative to C.T.). In a similar way, aV_L (augmented left arm) and aV_F (augmented left leg) leads are defined.

- Lead aV_R or "augmented vector right" has the positive electrode (white) on the right arm. The negative electrode is a combination of the left arm (black) electrode and the left leg (red) electrode, which "augments" the signal strength of the positive electrode on the right arm.

- Lead aV_L or "augmented vector left" has the positive (black) electrode on the left arm. The negative electrode is a combination of the right arm (white) electrode and the left leg (red) electrode, which "augments" the signal strength of the positive electrode on the left arm.

- Lead aV_F or "augmented vector foot" has the positive (red) electrode on the left leg. The negative electrode is a combination of the right arm (white) electrode and the left arm (black) electrode, which "augments" the signal of the positive electrode on the left leg

The augmented limb leads aV_R, aV_L, and aV_F are amplified in this way because the signal is too small to be useful when the negative electrode is Wilson's central terminal. Together with leads I, II, and III, augmented limb leads aV_R, aV_L, and aV_F form the basis of the hexaxial reference system, which is used to calculate the heart's electrical axis in the frontal plane.

3.2.3 Precordial leads

The precordial leads V1, V2, V3, V4, V5, and V6 are placed directly on the chest. Refer Figure 3.4 for Proper placement of the precordial leads.

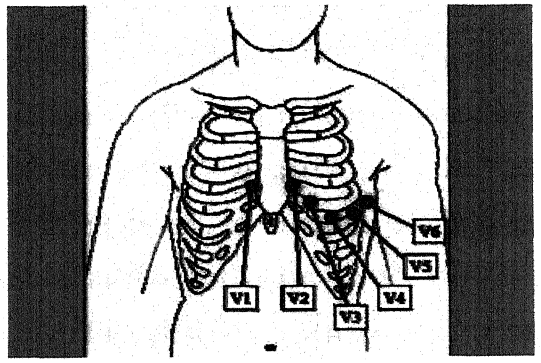


Fig. 3.4 :Proper placement of the precordial leads.

Because of their close proximity to the heart, they do not require augmentation. Wilson's central terminal is used for the negative electrode, and these leads are considered to be unipolar. The precordial leads view the heart's electrical activity [94] in the so-called horizontal plane. The heart's electrical axis in the horizontal plane is referred to as the Z axis

Six additional electrodes were introduced that are located in an array directly over the heart and hence, possibly, provide a greater sensitivity to devian source configurations. The signal of each precordial lead is measured between that lead and a common reference. The latter is called the Wilson central terminal (C.T.) and is formed by connecting each limb lead through the same value resistance, R, to a common point (the central terminal). If the recorder has a very high input impedance and if each electrode resistance is the same (or better yet, simply small compared with R), then since the total current entering the C.T. node considered algebraically, is zero. The C.T. potential will be the mean of the limb lead potentials. Wilson specified a resistance value of 5 kΩ, but this is too low; its loading will result in the C.T., failing to equal the mean

value of lead voltages if the electrode impedances are unequal (a normal occurane). In fact, to meet the required input impedance of 2.5 MΩ per lead (others grounded) of the ANSIAAMI [5], standard R should be chosen at least as large as 3.3 MΩ, which should also prove large compared with electrode-skin resistance.

Originally, it was thought that the C.T. was a true zero potential and hence an ideal reference. It is noted that electrocardiographic potentials are quasi-static, that is, they satisfy the equations of potential theory (Laplace and Poisson) at any instant. But it is clear from potential theory that any potential field can be defined only to within an arbitrary constant. So there is no true zero potential. On the other hand, one often chooses a reference electrode at a position that is remote from the sources. In these circumstances the resulting signal can be interpreted solely in terms of source-field behavior at the proximal (unipolar) lead. In contrast, for the bipolar case, the lead voltage depends on source-field behaviour at both electrodes. If leads are confined to the thorax, then no position is satisfactorily remote, but one can argue that the C.T. as an average of several peripheral potentials has some of the desired properties. In any case, the widespread use of the C.T. and the desire to describe ECG signals according to a standard method have perpetuated its Use. Following the argument that the C.T. behaves as a remote reference, one frequently sees the precordial leads referred to as unipolar. They are labeled V_1, V_2, V_3, V_4, V_5, and V_6 and have the following anatomical locations.

V_1 Fourth right intercostal space at the sternal edge

V_2 Fourth left intercostal space at the sternal edge

V_3 Midway between V_2 and V_4

V_4 Fifth left intercostal space at the midclavicular line

V_5 Same level as V_4 but at anterior axillary line

V_6 Same level as V_4 at midaxial line

Leads V1, V2, and V3 are referred to as the right precordial leads and V4, V5, and V6 are referred to as the left precordial leads.

The QRS complex should be negative in lead V1 and positive in lead V6. The QRS complex should show a gradual transition from negative to positive between leads

V2 and V4. The equiphasic lead is referred to as the transition lead. When the transition occurs earlier than lead V3, it is referred to as an early transition. When it occurs later than lead V3, it is referred to as a late transition. There should also be a gradual increase in the amplitude of the R wave between leads V1 and V4. This is known as R wave progression. Poor R wave progression is a nonspecific finding. It can be caused by conduction abnormalities, myocardial infarction, cardiomyopathy, and other pathological conditions.

3.2.4 Ground lead

An additional electrode (usually green) is present in modern four-lead and twelve-lead ECGs. This is the ground lead and is placed on the right leg by convention, although in theory it can be placed anywhere on the body. With a three-lead ECG, when one dipole is viewed, the remaining lead becomes the ground lead by default

3.2.5 Clinical lead groups

Therefore, there are twelve leads in total, each recording the electrical activity of the heart from a different perspective, which also correlates to a different anatomical area of the heart for the purpose of identifying acute coronary injury. Two leads that look at the same anatomical area of the heart are said to be contiguous. Refer Figure 3.5 for The Contiguous leads.

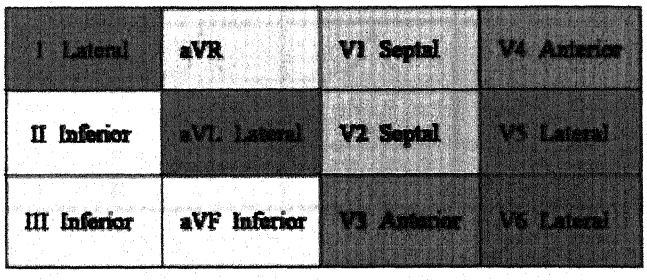


Fig. 3.5 : The Contiguous leads.

The inferior leads (leads II, III and aV_F) look at electrical activity from the vantage point of the inferior or diaphragmatic wall of the left ventricle.

- The lateral leads (I, aV_L, V_5 and V_6) look at the electrical activity from the vantage point of the lateral wall of left ventricle. Because the positive electrode

for leads I and aV_L are located on the left shoulder, leads I and aV_L are sometimes referred to as the high lateral leads. Because the positive electrodes for leads V5 and V6 are on the patient's chest, they are sometimes referred to as the low lateral leads.

- The septal leads, V_1 and V_2 look at electrical activity from the vantage point of the septal wall of the left ventricle. They are often grouped together with the anterior leads.
- The anterior leads, V_3 and V_4 look at electrical activity from the vantage point of the anterior wall of the left ventricle.
- In addition, any two precordial leads that are next to one another are considered to be contiguous. In other words, even though V4 is an anterior lead and V5 is a lateral lead, they are contiguous because they are next to one another.
- Lead aV_R offers no specific view of the left ventricle. Rather, it views the endocardial wall from its perspective on the right shoulder

Axis

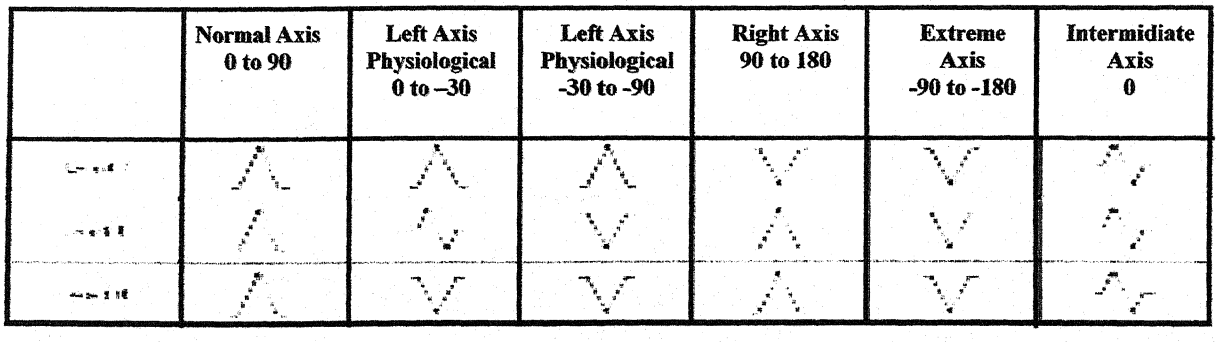


Fig. 3.6 : Diagram showing how the polarity of the QRS complex in leads I, II, and III can be used to estimate the heart's electrical axis in the frontal plane

The heart's electrical axis refers to the general direction of the heart's depolarization wavefront (or mean electrical vector) in the frontal plane. It is usually oriented in a right shoulder to left leg direction, which corresponds to the left inferior

quadrant of the hexaxial reference system , although -30° to +90° is considered to be normal. Figure 3.6 shows how the polarity of the QRS complex in leads I, II, and III can be used to estimate the heart's electrical axis in the frontal plane.

- Left axis deviation (-30° to -90°) may indicate left anterior fascicular block or Q waves from inferior MI Right axis deviation (+90° to +180°) may indicate left posterior fascicular block, Q waves from high lateral MI, or a right ventricular strain pattern.
- In the setting of right bundle branch block , right or left axis deviation may indicate bifascicular block

The set of 12 leads, namely V_I, V_{II}, V_{III}, V_1, V_2, V_3, V_4, V_5, V_6, aV_R, aV_L, aV_F, constitutes the standard 12-lead electrocardiogram [93, 94] that accounts for around 90% of all clinical electrocardiography. Figure 3.7 & 3.8 shows the types of lead connections with typical ECG waveform.

3.3 EXPERIMENTAL WORK

Lead II = (V_F- V_R) data is available and also it is obtained by the amplified analog signal of lead II on storage oscilloscope . The potentials at every one msec is measured and prepared a data table. Lead II signal is nothing but the difference of potentials between LL-RA i.e. (V_F-V_R) . Lead II data of more than 10 patients are calculated and average data for normal lead II is tabulated.

Augmented measurements provide the same wave shapes but 50% more potential output than the unused, non augmented unipolar limb lead measurements, V_R, V_L, V_F . The three unipolar leads bear a direct vector relationship to the three bipolar standard limb leads.

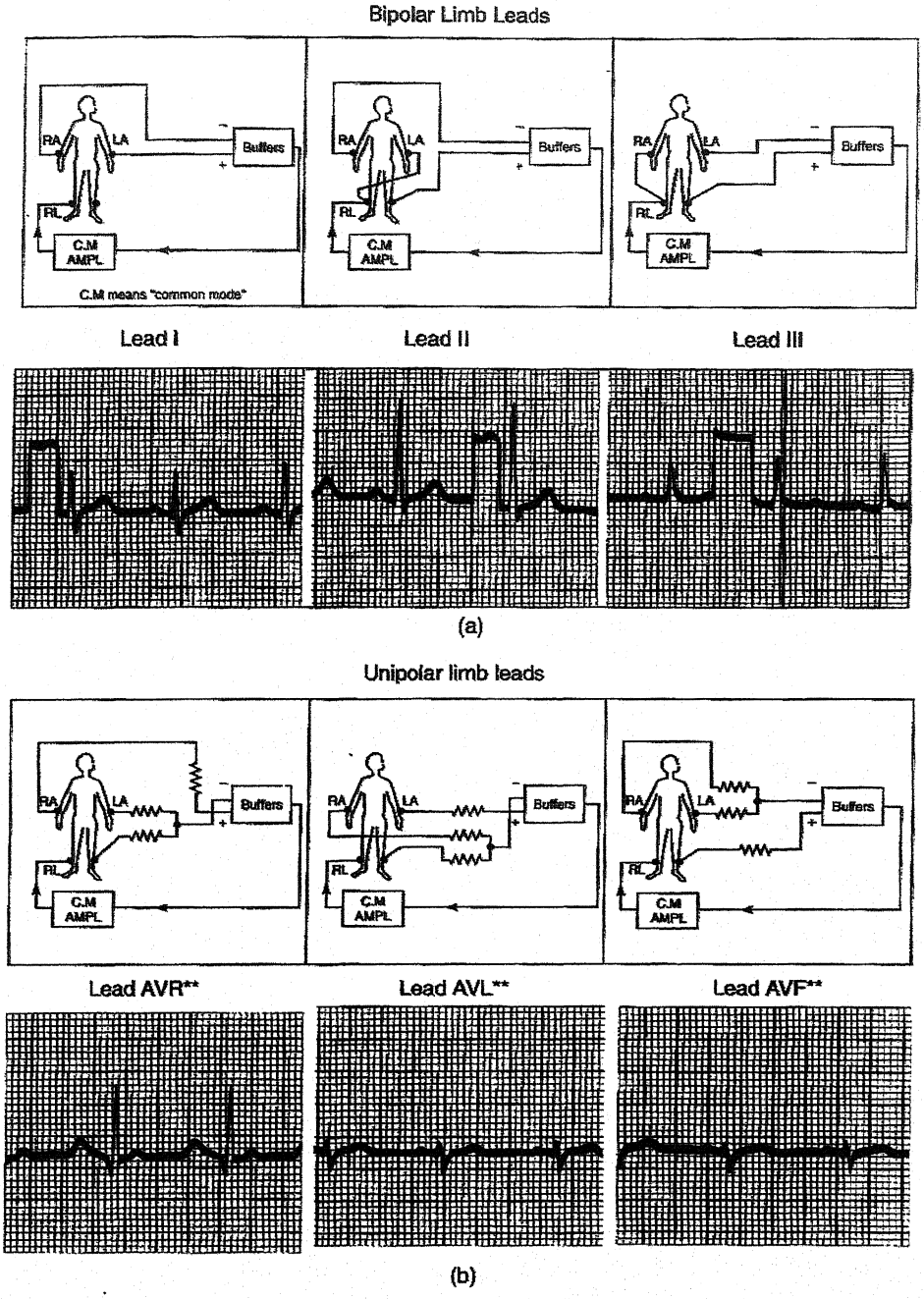


Fig. 3.7 : Types of lead connections with typical ECG waveform (a) bipolar timb leads (b) unipolar leads

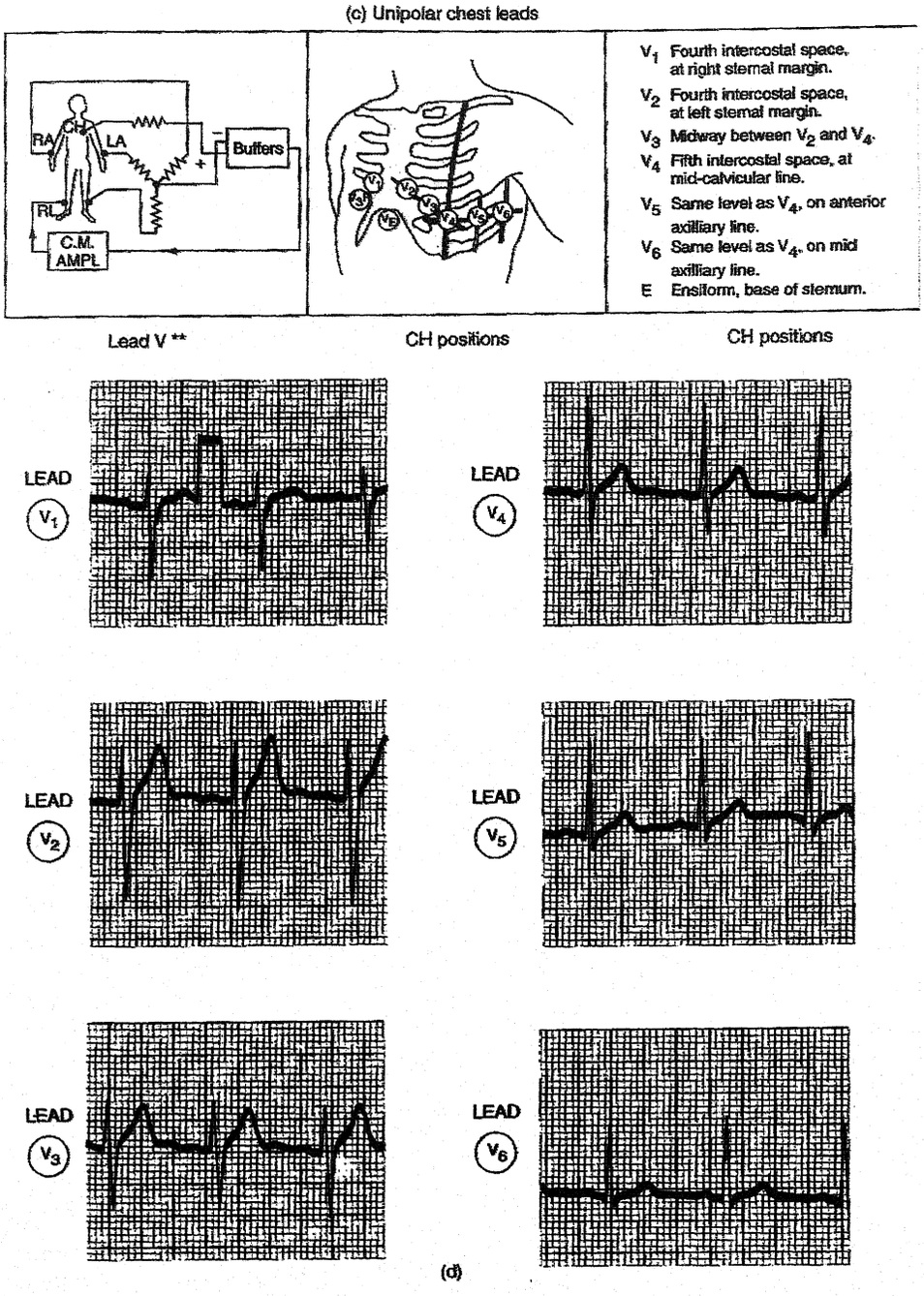


Fig. 3.8 : Types of lead connections with typical ECG waveform (c) position of the chest lead in unipolar precordial lead recording (d) C leads.

The aV_R unipolar measurement refers to the potential at the right arm using the left arm and left leg to form the in different electrode. The aV_L measurement refers to the potential at the left arm using both arms to form the in different electrode. The indifferent electrode is formed at the negative input of the amplifier in all cases. The ECG waveform is than be positive for aV_L, positive to aV_F but negative to aV_R. As

$$aV_R = (I + II)/2 \quad (26)$$

$$aV_R = -\ (V_L - V_R + V_F - V_R)/2 \quad (27)$$

$$aV_R = 3V_R/2 \quad (28)$$

$$aV_L = (I - III)/2 \quad (29)$$

$$\therefore \quad aV_L = (V_L - V_R - V_F + V_L)/2 \quad (30)$$

$$\therefore \quad aV_L = 3V_L/2 \quad (31)$$

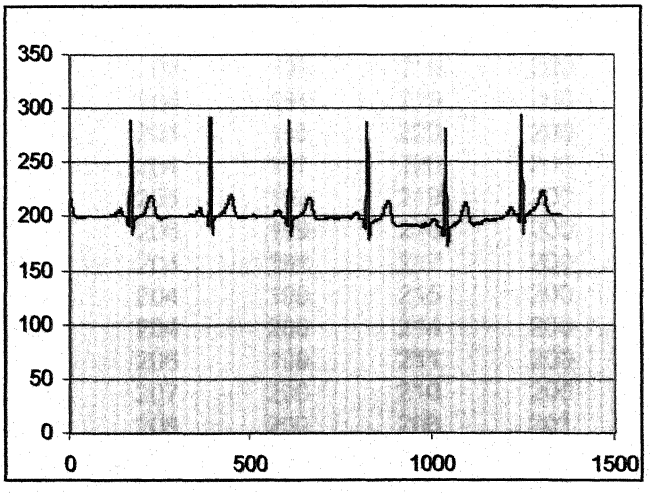
$$aV_F = (II + III)/2 \quad (32)$$

$$\therefore \quad aV_F = (V_L - V_R + V_F - V_L)/2 \quad (33)$$

$$\therefore \quad aV_F = 3V_F/2 \quad (34)$$

where $I = V_L - V_R$, $II = V_L - V_R$, $III = V_F - V_L$
and $V_R + V_L + V_F = 0$

based on these formulae the data is simulated for lead II signals for normal ECG and seventeen arrhythmias. These data is scaled to plot on a computer screen using Excel. The simulated data for each arrhythmia and its simulated lead II waveform is given as below :



HR = 68 beats /min.
X axis
500=2 sec.
Y axis
280=1.6 mV
200= 0 Volt

Fig. 3. 9 : Normal ECG lead II signal

1. NORMAL ECG SIGNAL

Refer figure 3. 9 for Normal ECG Lead II signal and table 3.1 for Normal ECG signal Lead II signal data :

Table 3.1 : Normal ECG lead II Data

220	200	201	205	200	202	201	202
218	200	201	204	200	201	201	202
217	200	200	202	201	200	200	201
215	201	200	202	201	199	200	201
213	200	200	201	202	199	200	201
211	200	201	200	202	199	200	201
209	200	201	200	201	199	200	202
207	200	200	201	202	199	200	201
206	200	201	200	202	199	200	202
204	200	201	200	203	199	200	201
203	200	201	200	202	199	200	201
202	200	201	200	203	199	200	202
201	201	200	200	202	199	200	202
201	200	201	200	203	198	200	203
200	200	201	201	204	199	200	204
200	200	201	200	204	198	200	204
200	200	201	198	204	198	200	205
200	200	202	195	206	199	200	206
199	200	202	192	206	199	200	206
200	200	201	192	207	199	201	205
199	200	201	197	208	199	200	205
199	200	201	209	209	199	200	205
199	200	201	230	210	199	200	206
199	200	201	253	211	200	200	207
199	200	202	273	212	200	201	207
199	200	201	290	214	200	201	208
199	200	201	290	214	200	200	207
200	200	202	248	215	199	200	208
199	201	201	216	216	200	201	207
199	201	201	198	218	199	200	206
199	200	201	184	218	199	201	205
199	200	202	183	219	200	200	203
199	200	202	190	219	200	200	202
200	200	204	194	219	200	200	202
199	200	204	195	220	200	200	202
199	200	204	197	220	200	201	201
200	200	205	198	219	200	201	200
199	201	206	199	218	200	201	201
199	200	204	198	217	200	201	201
199	200	204	199	215	200	201	200
199	200	204	200	214	200	201	201
200	200	206	199	211	200	201	200
199	201	207	200	210	200	201	200
200	201	206	200	208	201	201	201
200	200	206	199	206	200	201	200
200	200	207	200	205	201	201	199
200	201	208	200	203	200	201	196
200	201	206	200	203	201	201	192

192	212	200	200	199	202	198	199
196	212	200	200	199	203	198	199
207	213	200	200	199	204	198	199
226	214	201	200	199	204	198	199
250	216	200	201	199	205	198	199
270	216	200	200	199	206	198	199
287	218	200	200	199	206	198	199
292	218	200	200	198	207	198	199
254	218	200	200	194	208	198	199
220	219	201	200	191	209	198	199
200	220	200	200	191	210	198	199
186	221	201	200	196	211	199	199
183	221	200	200	209	212	198	199
190	220	200	201	231	213	198	199
195	218	200	200	254	214	198	199
196	217	200	201	273	215	199	199
197	216	201	200	290	216	199	199
199	214	201	201	286	216	198	199
200	212	201	200	244	218	199	199
199	210	201	200	214	218	199	199
200	209	201	200	195	218	199	199
200	207	202	201	184	218	199	199
200	205	200	200	182	218	199	200
200	204	200	200	190	218	199	199
201	202	200	200	194	216	199	199
201	202	200	200	195	215	200	199
201	200	200	201	196	213	199	199
201	201	200	201	197	212	199	199
201	200	199	203	198	210	199	199
201	200	200	203	198	208	199	199
201	200	201	203	198	206	199	198
201	199	200	204	199	204	199	199
202	199	200	205	199	202	199	199
201	200	200	205	199	201	199	199
202	199	200	204	199	201	198	200
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208	199	201	200	202	198	198	204
209	199	200	200	202	198	198	203
210	199	200	199	202	198	199	202
200	197	192	192	197	192	193	196

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198	197	192	192	196	191	193	197
198	198	192	192	196	191	192	197
198	198	192	193	196	193	193	197
197	198	192	192	194	193	193	196
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191	204	192	190	190	196	193	198
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188	206	193	190	190	197	194	198
192	207	193	189	189	198	194	198
202	208	192	191	189	199	194	198
222	208	192	193	190	200	194	198
246	210	193	192	187	201	194	198
265	211	193	191	183	202	194	198
283	212	192	190	182	203	194	198
288	212	193	191	185	204	193	198
250	214	192	192	198	206	194	198
216	214	193	194	216	206	195	199
196	214	192	194	240	207	196	198
182	214	192	194	260	209	194	199
179	214	192	193	278	210	196	199
184	214	192	192	282	210	196	199
190	213	192	192	243	212	196	199
191	211	193	193	209	212	196	199
193	210	192	192	189	213	196	199
194	207	193	193	174	213	197	199
194	206	192	193	172	213	197	199
195	203	192	193	178	212	197	200
195	202	192	192	182	211	197	200
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195	198	193	193	186	208	198	200
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196	195	192	195	190	202	197	202
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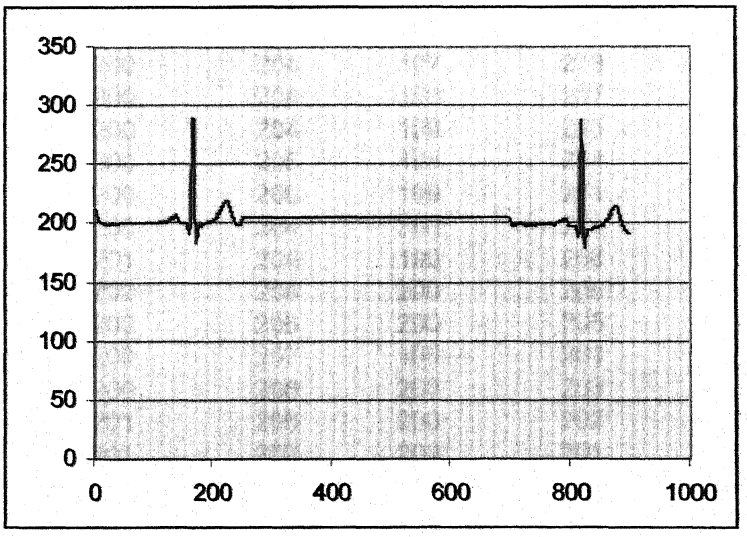
197	192	193	197	192	193	196	206
197	192	192	197	193	194	196	207
207	202	197	206	218	207	202	
207	203	200	206	219	206	202	

208	201	200	206	220	205	202
208	197	201	206	221	205	202
208	194	202	207	222	204	202
208	194	202	207	222	204	202
207	197	202	208	224	204	202
205	209	203	207	224	203	202
204	229	203	208	224	203	202
203	253	203	209	224	203	203
202	273	203	209	224	203	202
203	290	204	210	223	203	202
202	294	203	211	221	203	202
201	255	203	211	220	203	202
201	223	204	212	218	202	203
200	203	204	212	215	203	203
201	187	204	214	214	202	203
202	183	205	214	211	202	
202	190	205	216	209	202	
202	195	205	217	208	202	

2. BRADYCARDIA

Refer figure 3.10 for bradycardia lead II signal and table 3.2 for bradycardia lead II signal data.



HR = 30 beat/min.
X axis
200 = 600 msec.
Y axis'
200 = 0 V.
280 = 1.6 mV

Figure 3.10 : Bradycardia lead II wave.

Table 3.2 Bradycardia lead II data

213	200	201	200	202	205	205
211	200	201	200	203	205	205
209	200	201	200	204	205	205
207	200	201	200	204	205	205
206	200	200	201	204	205	205
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203	201	201	198	206	205	205
202	200	201	195	207	205	205
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201	200	202	192	209	205	205
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200	200	201	230	212	205	205
200	200	201	253	214	205	205
199	200	201	273	214	205	205
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205	205	205	205	205	205	205
205	205	205	205	205	205	205
205	205	205	205	205	205	204
205	205	205	205	205	205	203
205	205	205	205	205	205	202

205	205	205	205	205	205	201
205	205	205	205	205	205	200
205	205	205	205	205	205	199
205	205	205	205	205	205	198
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199	199	283	212	199	198	198
199	199	288	212	199	198	198
200	199	250	214	199	197	198
199	199	216	214	199	197	199
199	199	196	214	199	197	198
199	199	182	214	199	198	199
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199	201	195	200	199	192	207
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199	203	195	197	199	222	208
199	203	195	196	199	246	210
199	202	196	195			
199	203	196	194			
199	203	197	194			
198	204	196	193			
199	204	196	193			
199	204	196	192			
199	204	196	192			
198	204	196	192			
198	203	197	192			
199	202	197				
199	200	197				
199	199	197				

3. TACHYCARDIA

Refer figure 3.11 for Tachycardia lead II signal and table 3.3 for tachycardia lead II data.

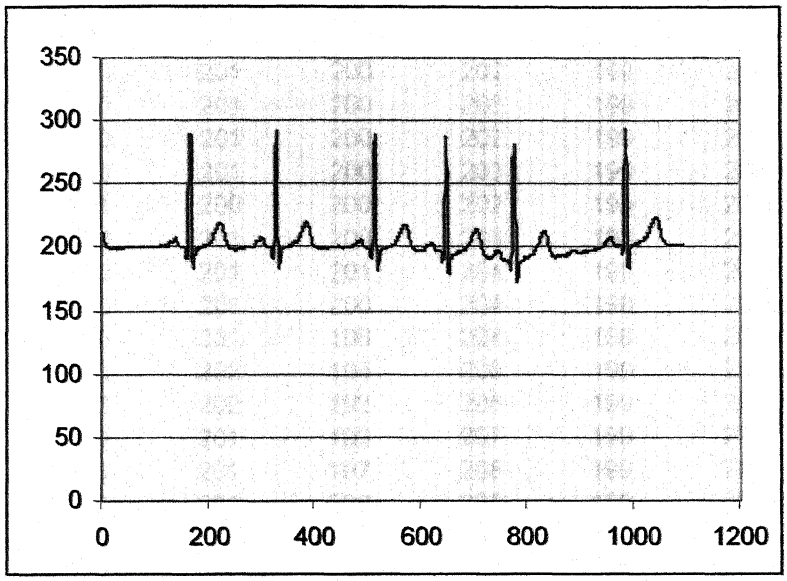

Fig. 3.11 : Tachycardia lead II signal

Table 3.3 : Tachycardia lead II data

213	200	200	201	202	199	204	195
211	200	201	200	202	199	204	196
209	200	201	200	201	199	205	197
207	200	200	201	202	199	206	199
206	200	201	200	202	199	206	200
204	200	201	200	203	199	205	199
203	200	201	200	202	199	205	200
202	200	201	200	203	199	205	200
201	201	200	200	202	199	206	200
201	200	201	200	203	198	207	200
200	200	201	201	204	199	207	201
200	200	201	200	204	198	208	201
200	200	201	198	204	198	207	201
200	200	202	195	206	199	208	201
199	200	202	192	206	199	207	201
200	200	201	192	207	199	206	201
199	200	201	197	208	199	205	201
199	200	201	209	209	199	203	201
199	200	201	230	210	199	202	202
199	200	201	253	211	200	202	201
199	200	202	273	212	200	202	202
199	200	201	290	214	200	201	203
199	200	201	290	214	200	200	202
200	200	202	248	215	199	201	203
199	201	201	216	216	200	201	203
199	201	201	198	218	199	200	204
199	200	201	184	218	199	201	204
199	200	202	183	219	200	200	205
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199	200	204	195	220	200	200	205
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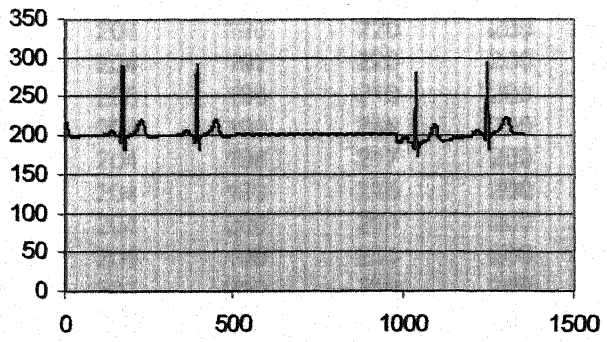
199	201	206	199	218	200	192	207
199	200	204	198	217	200	192	208
199	200	204	199	215	200	196	208
199	200	204	200	214	200	207	209
200	200	206	199	211	200	226	210
199	201	207	200	210	200	250	212
200	201	206	200	208	201	270	212
200	200	206	199	206	200	287	213
200	200	207	200	205	201	292	214
200	201	208	200	203	200	254	216
200	201	206	200	203	201	220	216
200	201	205	200	202	201	200	218
200	201	204	200	201	201	186	218
200	200	202	201	200	200	183	218
201	200	202	201	199	200	190	219

220	200	207	201	198	192	207	198
221	201	206	200	198	202	208	196
221	200	205	200	198	222	208	196
220	200	203	201	198	246	210	196
218	200	202	200	197	265	211	194
217	200	201	201	198	283	212	192
216	201	200	201	198	288	212	192
214	201	200	202	198	250	214	191
212	201	200	202	198	216	214	191
210	201	200	202	198	196	214	190
209	201	199	202	198	182	214	191
207	202	199	202	198	179	214	190
205	200	199	203	198	184	214	190
204	200	199	204	198	190	213	190
202	200	199	204	198	191	211	190
202	200	199	205	198	193	210	190
200	200	199	206	198	194	207	190
201	200	199	206	198	194	206	189
200	199	198	207	201	195	203	189
200	200	194	208	201	195	202	190
200	201	191	209	201	195	200	187
199	200	191	210	203	195	198	183
199	200	196	211	203	195	197	182
200	200	209	212	203	195	196	185
199	200	231	213	202	196	195	198
199	200	254	214	203	196	194	216
200	201	273	215	203	197	194	240
199	200	290	216	204	196	193	260
199	201	286	216	204	196	193	278
200	200	244	218	204	196	192	282
200	200	214	218	204	196	192	243
199	200	195	218	204	196	192	209
199	201	184	218	203	197	192	189
199	200	182	218	202	197	192	174
199	200	190	218	200	197	193	172
200	200	194	216	199	197	193	178
199	200	195	215	198	197	192	182
199	201	196	213	198	198	193	184
199	201	197	212	198	198	193	186
199	203	198	210	197	198	193	188
199	203	198	208	197	199	194	190
199	203	198	206	197	198	195	190
200	204	199	204	198	199	195	190
200	205	199	202	197	200	195	189
200	205	199	201	197	200	196	189
201	204	199	201	197	200	197	190
200	204	200	200	197	202	196	190
200	206	200	199	197	202	196	191
200	206	199	199	196	202	197	192
200	206	200	198	191	204	197	192
200	206	200	198	189	204	197	193
201	206	200	198	188	206	197	192

191	208	194	197	205	203	211	203
191	206	195	197	206	187	212	203
191	205	196	197	207	183	212	203
193	202	194	197	207	190	214	202
193	200	196	198	207	195	214	203
193	199	196	198	208	197	216	202
194	198	196	198	208	200	217	202
193	196	196	198	208	200	218	202
194	195	196	198	208	201	219	202
194	195	197	198	207	202	220	202
194	194	197	198	205	202	221	202
194	193	197	198	204	202	222	202
195	194	197	198	203	203	222	202
196	193	197	198	202	203	224	202
196	193	198	198	203	203	224	202
196	193	197	199	202	203	224	202
197	192	197	198	201	204	224	202
198	193	197	199	201	203	224	202
199	193	196	199	200	203	223	203
200	193	197	199	201	204	221	202
201	193	196	199	202	204	220	202
202	194	196	199	202	204	218	202
203	193	196	199	202	205	215	202
204	193	196	199	202	205	214	203
206	193	196	199	203	205	211	203
206	194	196	200	201	206	209	203
207	193	196	200	197	206	208	end
209	193	196	200	194	206	207	
210	193	196	200	194	206	206	
210	194	197	200	197	207	205	
212	194	197	200	209	207	205	
212	194	197	202	229	208	204	
213	194	196	203	253	207	204	
213	194	197	204	273	208	204	
213	194	197	203	290	209	203	
212	194	197	204	294	209	203	
211	194	197	206	255	210	203	
211	193	196	206	223	211	203	

4. MISSED BEAT



HR = 69beat/min.
X axis
200 = 600 msec.
Y axis'
200 = 0 V.
280 = 1.6 mV

Fig. 3.12 : Missed beat lead II signal

Refer figure 3.12 for missed beat lead II signal and table 3.4 for Missed beat data

Table 3.4 : Missed beat data

220	200	201	205	200	202	201	202
218	200	201	204	200	201	201	202
217	200	200	202	201	200	200	201
215	201	200	202	201	199	200	201
213	200	200	201	202	199	200	201
211	200	201	200	202	199	200	201
209	200	201	200	201	199	200	202
207	200	200	201	202	199	200	201
206	200	201	200	202	199	200	202
204	200	201	200	203	199	200	201
203	200	201	200	202	199	200	201
202	200	201	200	203	199	200	202
201	201	200	200	202	199	200	202
201	200	201	200	203	198	200	203
200	200	201	201	204	199	200	204
200	200	201	200	204	198	200	204
200	200	201	198	204	198	200	205
200	200	202	195	206	199	200	206
199	200	202	192	206	199	200	206
200	200	201	192	207	199	201	205
199	200	201	197	208	199	200	205
199	200	201	209	209	199	200	205
199	200	201	230	210	199	200	206
199	200	201	253	211	200	200	207
199	200	202	273	212	200	201	207
199	200	201	290	214	200	201	208
199	200	201	290	214	200	200	207
200	200	202	248	215	199	200	208
199	201	201	216	216	200	201	207
199	201	201	198	218	199	200	206
199	200	201	184	218	199	201	205
199	200	202	183	219	200	200	203
199	200	202	190	219	200	200	202
200	200	204	194	219	200	200	202
199	200	204	195	220	200	200	202
199	200	204	197	220	200	201	201
200	200	205	198	219	200	201	200
199	201	206	199	218	200	201	201
199	200	204	198	217	200	201	201
199	200	204	199	215	200	201	200
199	200	204	200	214	200	201	201
200	200	206	199	211	200	201	200
199	201	207	200	210	200	201	200
200	201	206	200	208	201	201	201
200	200	206	199	206	200	201	200
200	200	207	200	205	201	201	199
200	201	208	200	203	200	201	196
200	201	206	200	203	201	201	192

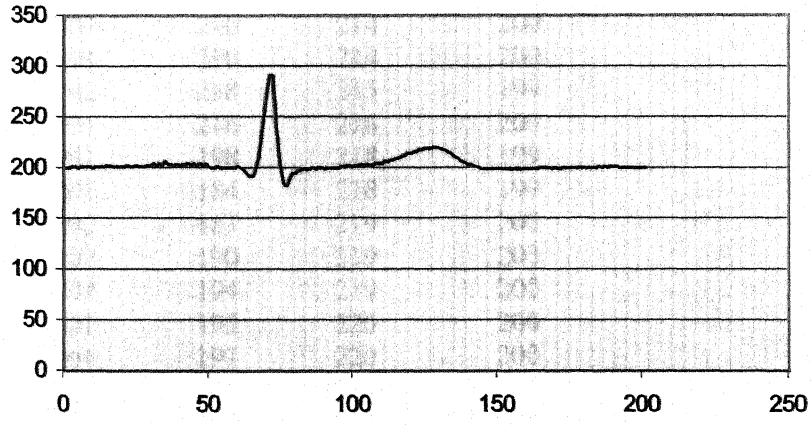
192	212	200	202	202	202	202	202
196	212	200	202	202	202	202	202
207	213	200	202	202	202	202	200
226	214	201	202	202	202	202	200
250	216	200	202	202	202	200	200
270	216	200	202	202	202	200	200
287	218	200	202	202	200	200	201
292	218	200	202	202	200	200	201
254	218	200	202	200	200	201	201
220	219	201	202	200	200	201	201
200	220	200	200	200	201	201	201
186	221	201	200	200	201	201	202
183	221	200	200	201	201	201	202
190	220	200	200	201	201	202	202
195	218	200	201	201	201	202	202
196	217	200	201	201	202	202	202
197	216	201	201	201	202	202	202
199	214	201	201	202	202	202	202
200	212	201	201	202	202	202	202
199	210	201	202	202	202	202	202
200	209	201	202	202	202	202	202
200	207	202	202	202	202	202	202
200	205	202	202	202	202	202	202
200	204	202	202	202	202	202	202
201	202	202	202	202	202	202	202
201	202	202	202	202	202	202	202
201	200	202	202	202	202	202	202
201	201	202	202	202	202	202	200
201	200	202	202	202	202	202	200
201	200	202	202	202	202	200	200
201	200	202	202	202	202	200	200
201	199	202	202	202	200	200	201
202	199	202	202	202	200	200	201
201	200	202	202	200	200	201	201
202	199	202	202	200	200	201	201
203	199	202	200	200	201	201	201
202	200	202	200	200	201	201	202
203	199	200	200	201	201	201	202
203	199	200	200	201	201	202	202
204	200	200	201	201	201	202	202
204	200	200	201	201	202	202	202
205	199	201	201	201	202	202	202
204	199	201	201	202	202	202	202
204	199	201	201	202	202	202	202
205	199	201	202	202	202	202	202
206	200	201	202	202	202	202	202
206	199	202	202	202	202	202	202
207	199	202	202	202	202	202	202
208	199	202	202	202	202	202	202
208	199	202	202	202	202	202	202
209	199	202	202	202	202	202	202
210	199	202	202	202	202	202	202

200	200	201	201	197	192	193	196
200	200	201	201	198	191	193	196
200	201	201	201	196	191	193	197
200	201	201	202	196	191	192	197
201	201	201	202	196	193	193	197
201	201	202	202	194	193	193	196
201	201	202	202	192	193	193	197
201	202	202	202	192	194	193	197
201	202	202	202	191	193	194	197
202	202	202	202	191	194	193	197
202	202	202	202	190	194	193	196
202	202	202	202	191	194	193	197
202	202	202	202	190	194	194	197
202	202	202	202	190	195	193	197
202	202	202	202	190	196	193	197
202	202	202	202	190	196	193	198
202	202	202	202	190	196	194	198
202	202	202	202	190	197	194	198
202	202	202	202	189	198	194	198
202	202	202	202	189	199	194	198
202	202	202	202	190	200	194	198
202	202	200	202	187	201	194	198
202	202	200	202	183	202	194	198
202	200	200	202	182	203	194	198
202	200	200	191	185	204	193	198
200	200	201	192	198	206	194	198
200	200	201	194	216	206	195	199
200	201	201	194	240	207	196	198
200	201	201	194	260	209	194	199
201	201	201	193	278	210	196	199
201	201	202	192	282	210	196	199
201	201	202	192	243	212	196	199
201	202	202	193	209	212	196	199
201	202	202	192	189	213	196	199
202	202	202	193	174	213	197	199
202	202	202	193	172	213	197	199
202	202	202	193	178	212	197	200
202	202	202	192	182	211	197	200
202	202	202	193	184	211	197	200
202	202	202	193	186	208	198	200
202	202	202	193	188	206	197	200
202	202	202	194	190	205	197	200
202	202	202	195	190	202	197	202
202	202	202	195	190	200	196	203
202	202	202	195	189	199	197	204
202	202	202	196	189	198	196	203
202	202	200	197	190	196	196	204
202	202	200	196	190	195	196	206
202	200	200	196	191	195	196	206
202	200	200	197	192	194	196	205
200	200	201	197	192	193	196	206
200	200	201	197	193	194	196	207
207	202	197	206	218	207	202	
207	203	200	206	219	206	202	
208	201	200	206	220	205	202	

208	197	201	206	221	205	202
208	194	202	207	222	204	202
208	194	202	207	222	204	202
207	197	202	208	224	204	202
205	209	203	207	224	203	202
204	229	203	208	224	203	202
203	253	203	209	224	203	203
202	273	203	209	224	203	202
203	290	204	210	223	203	202
202	294	203	211	221	203	202
201	255	203	211	220	203	202
201	223	204	212	218	202	203
200	203	204	212	215	203	203
201	187	204	214	214	202	203
202	183	205	214	211	202	
202	190	205	216	209	202	
202	195	205	217	208	202	

5. ATRIAL FIBRILLATION

Refer figure 3.13 for atrial fibrillation lead II signal and table 3.5 for atrial fibrillation data



HR = 68 beat/min.
X axis
250 = 1 sec.
Y axis'
200 = 0 V.
280 = 1.6 mV

Fig. 3.13 : Atrial fibrillation lead II signal

Table 3.5 Atrial fibrillation data

200	204	201	199	200
200	201	202	199	200
201	200	202	199	200
201	200	201	199	200
200	201	202	199	200
201	200	202	199	200
201	200	203	199	200
201	200	202	199	200
201	200	203	199	200
200	200	202	199	
201	200	203	198	
201	201	204	199	
201	200	204	198	
201	198	204	198	
202	195	206	199	
202	192	206	199	
201	192	207	199	
201	197	208	199	
201	209	209	199	
201	230	210	199	
201	253	211	200	
202	273	212	200	
201	290	214	200	
201	290	214	200	
202	248	215	199	
201	216	216	200	
201	198	218	199	
201	184	218	199	
202	183	219	200	
202	190	219	200	
204	194	219	200	
202	195	220	200	
204	197	220	200	
202	198	219	200	
206	199	218	200	
203	198	217	200	
203	199	215	200	
202	200	214	200	
203	199	211	200	
202	200	210	200	
203	200	208	201	
203	199	206	200	
204	200	205	201	
202	200	203	200	
204	200	203	201	
202	200	202	201	
204	200	201	201	
202	201	200	200	

6 VENTRICULAR FIBRILLATION

Table 3.6 : Ventricular Fibrillation data

220	200	201	205	200	202	201	202
218	200	201	204	200	201	201	202
217	200	200	202	201	200	200	201
215	201	200	202	201	199	200	201
213	200	200	201	202	199	200	201
211	200	201	200	202	199	200	201
209	200	201	200	201	199	200	202
207	200	200	201	202	199	200	201
206	200	201	200	202	199	200	202
204	200	201	200	203	199	200	201
203	200	201	200	202	199	200	201
202	250	201	200	203	199	200	202
201	201	200	200	202	199	200	202
201	170	201	200	203	198	200	203
200	165	201	201	204	250	200	204
200	160	201	200	204	240	200	204
200	155	201	198	204	235	200	205
200	150	202	195	206	230	200	206
199	160	202	192	206	225	200	206
200	165	201	192	207	220	201	205
199	170	201	197	208	215	200	205
199	175	201	209	209	210	200	205
199	180	201	200	210	199	200	206
199	185	201	190	211	200	200	207
199	190	202	180	212	200	201	207
199	195	201	170	214	200	201	208
199	195	201	190	214	200	200	207
200	195	202	180	215	199	200	208
199	195	201	170	216	200	201	207
199	201	201	198	218	199	200	206
199	200	201	184	218	199	201	205
199	200	202	183	219	200	200	203
199	200	202	190	219	200	200	202
200	200	204	194	219	200	200	202
199	200	204	195	220	200	200	202
199	200	204	197	220	200	201	201
200	200	205	198	219	200	201	200
199	201	206	199	218	200	201	201
240	200	204	198	217	200	201	201
235	200	204	199	215	200	201	200
230	200	204	200	214	200	201	201
225	200	206	199	211	200	201	200
229	201	207	200	210	200	201	200
200	201	206	200	208	201	201	201
200	200	206	199	206	200	201	200
200	200	207	200	205	201	201	199
200	201	208	200	203	200	201	196
200	201	206	200	203	201	201	192

192	212	200	200	230	202	198	199
196	212	200	200	190	203	198	199
200	213	200	200	150	204	198	199
190	214	201	200	199	204	198	199
180	216	200	201	199	205	198	199
170	216	200	200	199	206	198	199
160	218	200	200	199	206	198	199
150	218	200	200	198	207	198	199
170	218	200	200	194	208	198	199
180	219	201	200	191	209	198	199
190	220	200	200	191	210	198	199
186	221	201	200	196	211	199	199
183	221	200	200	209	212	198	199
190	220	200	201	231	213	198	199
195	218	200	200	220	214	198	199
196	217	200	201	210	215	199	199
197	216	201	200	240	216	199	199
199	214	201	201	230	216	198	199
200	212	201	200	230	218	230	199
199	210	201	200	214	218	220	199
200	209	201	200	195	218	190	199
200	207	202	201	184	218	150	199
200	205	200	200	182	218	140	200
200	204	200	200	190	218	130	199
201	202	200	200	194	216	160	199
201	202	200	200	195	215	140	199
201	200	200	201	196	213	145	199
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201	200	199	203	198	210	160	199
201	200	200	203	198	208	165	199
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201	199	200	204	199	204	175	199
202	199	200	205	199	202	180	199
201	200	200	205	199	201	185	199
202	199	200	204	199	201	190	200
203	199	200	204	200	200	195	200
202	200	200	206	200	199	220	201
203	199	200	206	260	199	230	201
203	199	200	206	270	198	225	201
204	200	200	206	200	198	235	203
204	200	201	206	200	198	230	203
205	199	201	207	201	198	225	203
204	199	201	206	200	198	225	202
204	199	200	205	200	198	199	203
205	199	200	203	201	198	199	203
206	200	200	202	200	197	198	204
206	199	201	201	201	198	199	204
207	199	201	200	201	198	199	204
208	199	200	200	202	198	199	204
208	199	201	200	202	198	198	204
209	199	200	200	202	198	198	203
210	199	200	220	202	198	199	end

Refer figure 3.14 for Ventricular fibrillation lead II signal and table 3.6 for ventricular fibrillation data

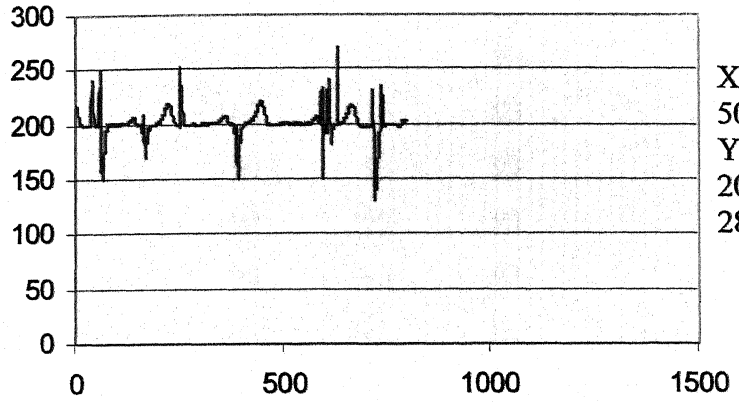

Fig. 3.14 : Ventricular fibrillation lead II signal

7. BIGEMINY

Refer figure 3.15 for Bigeminy lead II signal and table 3.7 for Bigeminy data

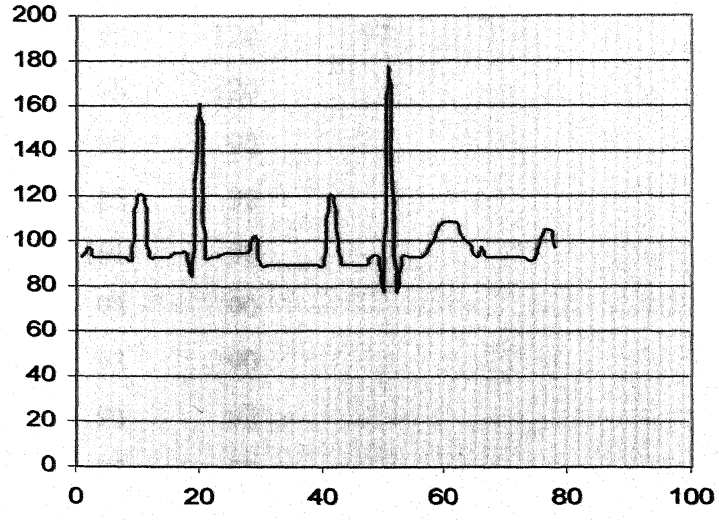

Fig. 3.15 : Bigeminy lead II signal

Table 3. 7 : Bigeminy data

93	95	177	93
97	95	79	93
93	95	93	93
93	103	93	93
93	90	93	93
93	90	93	93
93	90	95	93
93	90	101	91
91	90	107	95
120	90	109	103
120	90	109	105
93	90	109	97
93	90	101	
93	90	99	
93	90	93	
95	120	97	
95	120		
95	90		
85	90		
161	90		
93	90		
93	90		
93	93		
95	93		
95	79		

8. COUPLET

Table 3.8 Couplet data

220	200	201	205	200	202	201	202
218	200	201	204	200	201	201	202
217	200	200	202	201	200	200	201
215	201	200	202	201	199	200	201
213	200	200	201	202	199	200	201
211	200	201	200	202	199	200	201
209	200	201	200	201	199	200	202
207	200	200	201	202	199	200	201
206	200	201	200	202	199	200	202
204	200	201	200	203	199	200	201
203	200	201	200	202	199	200	201
202	200	201	200	203	199	200	202
201	201	200	200	202	199	200	202
201	200	201	200	203	198	200	203
200	200	201	201	204	199	200	204
200	200	201	200	204	198	200	204
200	200	201	198	204	198	200	205
200	200	202	195	206	199	200	206
199	200	202	192	206	199	200	206
200	200	201	192	207	199	201	205
199	200	201	197	208	199	200	205
199	200	201	209	209	199	200	205
199	200	201	230	210	199	200	206
199	200	201	253	211	200	200	207
199	200	202	273	212	200	201	207
199	200	201	290	214	200	201	208
199	200	201	290	214	200	200	207
200	200	202	248	215	199	200	208
199	201	201	216	216	200	201	207
199	201	201	198	218	199	200	206
199	200	201	184	218	199	201	205
199	200	202	183	219	200	200	203
199	200	202	190	219	200	200	202
200	200	204	194	219	200	200	202
199	200	204	195	220	200	200	202
199	200	204	197	220	200	201	201
200	200	205	198	219	200	201	200
199	201	206	199	218	200	201	201
199	200	204	198	217	200	201	201
199	200	204	199	215	200	201	200
199	200	204	200	214	200	201	201
200	200	206	199	211	200	201	200
199	201	207	200	210	200	201	200
200	201	206	200	208	201	201	201
200	200	206	199	206	200	201	200
200	200	207	200	205	201	201	199
200	201	208	200	203	200	201	196
200	201	206	200	203	201	201	192

192	212	200	200	199	202	140	199
196	212	200	200	199	203	135	199
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226	214	201	200	199	204	130	199
250	216	200	201	199	205	125	199
270	216	200	200	199	206	130	199
287	218	200	200	199	206	135	199
292	218	200	200	198	207	140	199
254	218	200	200	194	208	145	199
220	219	201	200	191	209	150	199
200	220	200	200	191	210	155	199
186	221	201	200	196	211	160	199
183	221	200	200	190	212	165	199
190	220	200	201	180	213	170	199
195	218	200	200	180	214	170	199
196	217	200	201	170	215	175	199
197	216	201	200	170	216	180	199
199	214	201	201	160	216	185	199
200	212	201	200	160	218	190	199
199	210	201	200	150	218	190	199
200	209	201	200	150	218	195	199
200	207	202	201	140	218	195	199
200	205	200	200	140	218	199	200
200	204	200	200	130	218	199	199
201	202	200	200	130	216	199	199
201	202	200	200	125	215	200	199
201	200	200	201	130	213	199	199
201	201	200	201	130	212	199	199
201	200	199	203	130	210	199	199
201	200	200	203	135	208	199	199
201	200	201	203	135	206	199	198
201	199	200	204	140	204	199	199
202	199	200	205	145	202	199	199
201	200	200	205	150	201	199	199
202	199	200	204	155	201	198	200
203	199	200	204	160	200	199	200
202	200	200	206	165	199	199	201
203	199	200	206	170	199	199	201
203	199	200	206	175	195	199	201
204	200	200	206	175	195	198	203
204	200	201	206	180	190	199	203
205	199	201	207	180	190	199	203
204	199	201	206	185	185	199	202
204	199	200	205	185	180	199	203
205	199	200	203	201	175	199	203
206	200	200	202	200	170	198	204
206	199	201	201	201	170	199	204
207	199	201	200	201	165	199	204
208	199	200	200	202	160	199	204
208	199	201	200	202	155	198	204
209	199	200	200	202	150	198	203
210	199	200	199	202	145	199	202

200	197	192	192	197	192	193	196
199	197	192	192	198	191	193	196
198	197	192	192	196	191	193	197
198	198	192	192	196	191	192	197
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197	200	192	191	191	194	193	197
197	200	192	191	190	194	193	196
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202	208	192	191	189	199	194	198
222	208	192	193	190	200	194	198
246	210	193	192	187	201	194	198
265	211	193	191	183	202	194	198
283	212	192	190	182	203	194	198
288	212	193	191	185	204	193	198
250	214	192	192	198	206	194	198
216	214	193	194	216	206	195	199
196	214	192	194	240	207	196	198
182	214	192	194	260	209	194	199
179	214	192	193	278	210	196	199
184	214	192	192	282	210	196	199
190	213	192	192	243	212	196	199
191	211	193	193	209	212	196	199
193	210	192	192	189	213	196	199
194	207	193	193	174	213	197	199
194	206	192	193	172	213	197	199
195	203	192	193	178	212	197	200
195	202	192	192	182	211	197	200
195	200	192	193	184	211	197	200
195	198	193	193	186	208	198	200
195	197	192	193	188	206	197	200
195	196	193	194	190	205	197	200
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196	194	193	195	190	200	196	203
197	194	192	195	189	199	197	204
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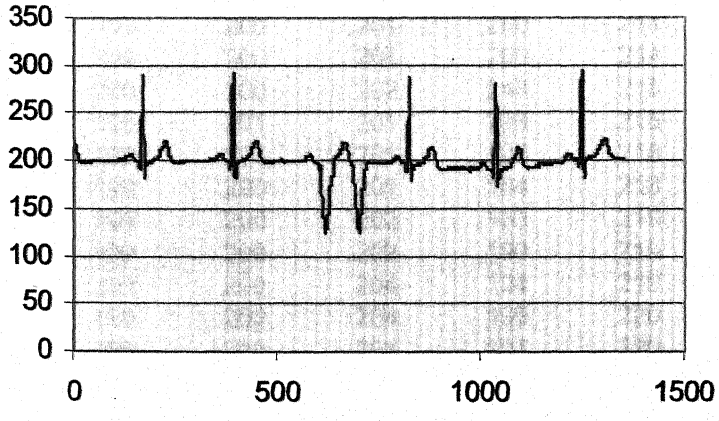
196	193	192	197	190	196	196	204
196	192	192	196	190	195	196	206
196	192	192	196	191	195	196	206
196	192	192	197	192	194	196	205
197	192	193	197	192	193	196	206
197	192	192	197	193	194	196	207

207	194	203	211	214	202
207	194	203	211	211	202
208	197	203	212	209	202
208	209	204	212	208	202
208	229	203	214	207	202
208	253	203	214	206	202
207	273	204	216	205	202
205	290	204	217	205	202
204	294	204	218	204	202
203	255	205	219	204	203
202	223	205	220	204	202
203	203	205	221	203	202
202	187	206	222	203	202
201	183	206	222	203	202
201	190	206	224	203	203
200	195	206	224	203	203
201	197	207	224	203	203
202	200	207	224	203	
202	200	208	224	202	
202	201	207	223	203	
202	202	208	221	202	
203	202	209	220	202	
201	202	209	218	202	
197	203	210	215	202	

Refer figure 3.16 for Couplet lead II signal and table 3.8 for couplet data



HR = 68 beats/min
X axis
500= 2 sec.
Y axis'
200 = 0 V.
280 = 1.6 mV

Fig. 3.16 : Couplet lead II signal

9. RUN

Table 3.9 : Run data

220	200	201	205	200	202	201	202
218	200	201	204	200	201	201	202
217	200	200	202	201	200	200	201
215	201	200	202	201	199	200	201
213	200	200	201	202	199	200	201
211	200	201	200	202	199	200	201
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200	200	202	195	206	199	200	206
199	200	202	192	206	199	200	206
200	200	201	192	207	199	201	205
199	200	201	197	208	199	200	205
199	200	201	209	209	199	200	205
199	200	201	230	210	199	200	206
199	200	201	253	211	200	200	207
199	200	202	273	212	200	201	207
199	200	201	290	214	200	201	208
199	200	201	290	214	200	200	207
200	200	202	248	215	199	200	208
199	201	201	216	216	200	201	207
199	201	201	198	218	199	200	206
199	200	201	184	218	199	201	205
199	200	202	183	219	200	200	203
199	200	202	190	219	200	200	202
200	200	204	194	219	200	200	202
199	200	204	195	220	200	200	202
199	200	204	197	220	200	201	201
200	200	205	198	219	200	201	200
199	201	206	199	218	200	201	201
199	200	204	198	217	200	201	201
199	200	204	199	215	200	201	200
199	200	204	200	214	200	201	201
200	200	206	199	211	200	201	200
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200	201	206	200	208	201	201	201
200	200	206	199	206	200	201	200
200	200	207	200	205	201	201	199
200	201	208	200	203	200	201	196
200	201	206	200	203	201	201	192

192	212	200	200	199	202	140	199
196	212	200	200	199	203	135	199
207	213	200	200	199	204	130	199
226	214	201	200	199	204	130	199
250	216	200	201	199	205	125	199
270	216	200	200	199	206	130	199
287	218	200	200	199	206	135	199
292	218	200	200	198	207	140	199
254	218	200	200	194	208	145	199
220	219	201	200	191	209	150	190
200	220	200	200	191	210	155	185
186	221	201	200	196	211	160	180
183	221	200	200	190	212	165	170
190	220	200	201	180	213	170	170
195	218	200	200	180	214	170	165
196	217	200	201	170	215	175	160
197	216	201	200	170	216	180	150
199	214	201	201	160	216	185	140
200	212	201	200	160	218	190	135
199	210	201	200	150	218	190	130
200	209	201	200	150	218	195	125
200	207	202	201	140	218	195	130
200	205	200	200	140	218	199	135
200	204	200	200	130	218	199	140
201	202	200	200	130	216	199	145
201	202	200	200	125	215	200	150
201	200	200	201	130	213	205	160
201	201	200	201	130	212	207	165
201	200	199	203	130	210	210	170
201	200	200	203	135	208	215	170
201	200	201	203	135	206	220	175
201	199	200	204	140	204	220	180
202	199	200	205	145	202	220	185
201	200	200	205	150	201	215	190
202	199	200	204	155	201	215	190
203	199	200	204	160	200	210	200
202	200	200	206	165	199	210	201
203	199	200	206	170	199	205	201
203	199	200	206	175	195	205	201
204	200	200	206	175	195	202	203
204	200	201	206	180	190	202	203
205	199	201	207	180	190	202	203
204	199	201	206	185	185	199	202
204	199	200	205	185	180	199	203
205	199	200	203	201	175	199	203
206	200	200	202	200	170	198	204
206	199	201	201	201	170	199	204
207	199	201	200	201	165	199	204
208	199	200	200	202	160	199	204
208	199	201	200	202	155	198	204
209	199	200	200	202	150	198	203
210	199	200	199	202	145	199	202

200	195	192	192	197	192	193	196
199	200	192	192	198	191	193	196
198	200	192	192	196	191	193	197
198	198	192	192	196	191	192	197
198	198	192	193	196	193	193	197
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197	200	192	191	191	194	193	197
197	200	192	191	190	194	193	196
197	200	192	191	191	194	193	197
197	202	192	191	190	194	194	197
197	202	192	192	190	195	193	197
196	202	192	192	190	196	193	197
191	204	192	190	190	196	193	198
189	204	192	192	190	196	194	198
188	206	193	190	190	197	194	198
192	207	193	189	189	198	194	198
202	208	192	191	189	199	194	198
222	208	192	193	190	200	194	198
222	210	193	192	187	201	194	198
220	211	193	191	183	202	194	198
210	212	192	190	182	203	194	198
210	212	193	191	185	204	193	198
205	214	192	192	198	206	194	198
205	214	193	194	216	206	195	199
200	214	192	194	240	207	196	198
200	214	192	194	260	209	194	199
190	214	192	193	278	210	196	199
185	214	192	192	282	210	196	199
186	213	192	192	243	212	196	199
180	211	193	193	209	212	196	199
170	210	192	192	189	213	196	199
160	207	193	193	174	213	197	199
150	206	192	193	172	213	197	199
140	203	192	193	178	212	197	200
130	202	192	192	182	211	197	200
125	200	192	193	184	211	197	200
130	198	193	193	186	208	198	200
135	197	192	193	188	206	197	200
140	196	193	194	190	205	197	200
145	195	192	195	190	202	197	202
145	194	193	195	190	200	196	203
150	194	192	195	189	199	197	204
160	193	192	196	189	198	196	203
165	193	192	197	190	196	196	204
170	192	192	196	190	195	196	206
175	192	192	196	191	195	196	206
180	192	192	197	192	194	196	205
190	192	193	197	192	193	196	206
190	192	192	197	193	194	196	207

207	202	197	206	218	207	202
207	203	200	206	219	206	202
208	201	200	206	220	205	202
208	197	201	206	221	205	202
208	194	202	207	222	204	202
208	194	202	207	222	204	202
207	197	202	208	224	204	202
205	209	203	207	224	203	202
204	229	203	208	224	203	202
203	253	203	209	224	203	203
202	273	203	209	224	203	202
203	290	204	210	223	203	202
202	294	203	211	221	203	202
201	255	203	211	220	203	202
201	223	204	212	218	202	203
200	203	204	212	215	203	203
201	187	204	214	214	202	203
202	183	205	214	211	202	end
202	190	205	216	209	202	
202	195	205	217	208	202	

Refer figure 3.17 for Run lead II signal and table 3.9 for run data

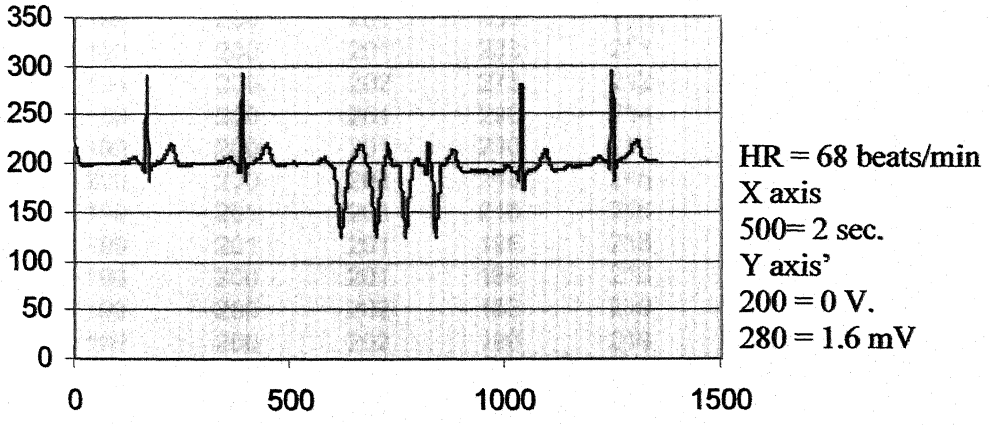

Fig. 3.17 : Run lead II signal

10. MULTIFOCAL RUN

Table 3.10 : Multifocal Run data

220	200	201	205	200	202	201	202
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217	200	200	202	201	200	200	201
215	201	200	202	201	199	200	201
213	200	200	201	202	199	200	201
211	200	201	200	202	199	200	201
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207	200	200	201	202	199	200	201
206	200	201	200	202	199	200	202
204	200	201	200	203	199	200	201
203	200	201	200	202	199	200	201
202	200	201	200	203	199	200	202
201	201	200	200	202	199	200	202
201	200	201	200	203	198	200	203
200	200	201	201	204	199	200	204
200	200	201	200	204	198	200	204
200	200	201	198	204	198	200	205
200	200	202	195	206	199	200	206
199	200	202	192	206	199	200	206
200	200	201	192	207	199	201	205
199	200	201	197	208	199	200	205
199	200	201	209	209	199	200	205
199	200	201	230	210	199	200	206
199	200	201	253	211	200	200	207
199	200	202	273	212	200	201	207
199	200	201	290	214	200	201	208
199	200	201	290	214	200	200	207
200	200	202	248	215	199	200	208
199	201	201	216	216	200	201	207
199	201	201	198	218	199	200	206
199	200	201	184	218	199	201	205
199	200	202	183	219	200	200	203
199	200	202	190	219	200	200	202
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199	200	204	195	220	200	200	202
199	200	204	197	220	200	201	201
200	200	205	198	219	200	201	200
199	201	206	199	218	200	201	201
199	200	204	198	217	200	201	201
199	200	204	199	215	200	201	200
199	200	204	200	214	200	201	201
200	200	206	199	211	200	201	200
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200	201	206	200	208	201	201	201
200	200	206	199	206	200	201	200
200	200	207	200	205	201	201	199
200	201	208	200	203	200	201	196
200	201	206	200	203	201	201	192

192	212	200	200	199	202	198	199
186	212	200	200	199	203	198	199
176	213	200	200	199	204	198	199
166	214	201	200	199	204	198	199
150	216	200	201	199	205	198	199
130	216	200	200	199	206	198	199
110	218	200	200	199	206	198	199
110	218	200	200	198	207	198	199
125	218	200	200	194	208	198	199
125	219	201	200	191	209	198	199
125	220	200	200	191	210	198	199
125	221	201	200	196	211	199	199
140	221	200	200	209	212	198	199
140	220	198	201	231	213	198	199
140	218	198	200	254	214	198	199
150	217	195	201	273	215	199	199
150	216	195	200	290	216	199	199
150	214	190	201	286	216	198	199
150	212	190	200	286	218	199	199
150	210	185	200	286	218	199	199
160	209	185	200	286	218	199	199
160	207	180	201	286	218	199	199
170	205	180	200	250	218	199	200
170	204	175	200	250	218	199	199
180	202	175	200	250	216	199	199
201	202	170	200	250	215	200	199
201	200	170	201	196	213	199	199
201	201	165	201	195	212	199	199
201	200	165	203	190	210	199	199
201	200	160	203	185	208	199	199
201	200	160	203	185	206	199	198
201	199	150	204	180	204	199	199
202	199	150	205	180	202	199	199
201	200	140	205	170	201	199	199
202	199	140	204	170	201	198	200
203	199	200	204	170	200	199	200
202	200	200	206	180	199	199	201
203	199	200	206	180	199	199	201
203	199	200	206	190	198	199	201
204	200	200	206	190	198	198	203
204	200	201	206	200	198	199	203
205	199	201	207	201	198	199	203
204	199	201	206	200	198	199	202
204	199	200	205	200	198	199	203
205	199	200	203	201	198	199	203
206	200	200	202	200	197	198	204
206	199	201	201	201	198	199	204
207	199	201	200	201	198	199	204
208	199	200	200	202	198	199	204
208	199	201	200	202	198	198	204
209	199	200	200	202	198	198	203
210	199	200	199	202	198	199	202

200	197	192	192	197	192	193	196
199	197	192	192	198	191	193	196
198	197	192	192	196	191	193	197
198	198	192	192	196	191	192	197
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189	204	192	192	190	196	194	198
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222	208	192	193	190	200	194	198
246	210	193	192	187	201	194	198
265	211	193	191	183	202	194	198
283	212	192	190	182	203	194	198
288	212	193	191	185	204	193	198
250	214	192	192	198	206	194	198
216	214	193	194	216	206	195	199
196	214	192	194	240	207	196	198
182	214	192	194	260	209	194	199
179	214	192	193	278	210	196	199
184	214	192	192	282	210	196	199
190	213	192	192	243	212	196	199
191	211	193	193	209	212	196	199
193	210	192	192	189	213	196	199
194	207	193	193	174	213	197	199
194	206	192	193	172	213	197	199
195	203	192	193	178	212	197	200
195	202	192	192	182	211	197	200
195	200	192	193	184	211	197	200
195	198	193	193	186	208	198	200
195	197	192	193	188	206	197	200
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196	195	192	195	190	202	197	202
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197	194	192	195	189	199	197	204
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196	193	192	197	190	196	196	204
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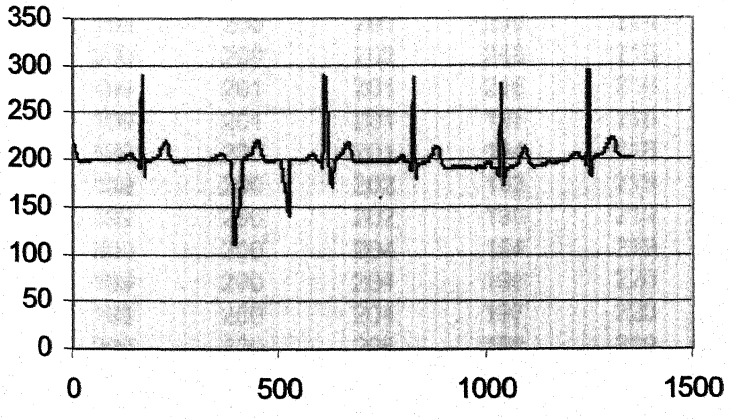
196	192	192	196	191	195	196	206
196	192	192	197	192	194	196	205
197	192	193	197	192	193	196	206
197	192	192	197	193	194	196	207

207	202	197	206	218	207	202
207	203	200	206	219	206	202
208	201	200	206	220	205	202
208	197	201	206	221	205	202
208	194	202	207	222	204	202
208	194	202	207	222	204	202
207	197	202	208	224	204	202
205	209	203	207	224	203	202
204	229	203	208	224	203	202
203	253	203	209	224	203	203
202	273	203	209	224	203	202
203	290	204	210	223	203	202
202	294	203	211	221	203	202
201	255	203	211	220	203	202
201	223	204	212	218	202	203
200	203	204	212	215	203	203
201	187	204	214	214	202	203
202	183	205	214	211	202	end
202	190	205	216	209	202	
202	195	205	217	208	202	

Refer figure 3.18 for Multifocal run lead II signal and table 3.10 for multifocal run data



HR = 68 beats/min
X axis
500= 2 sec.
Y axis'
200 = 0 V.
280 = 1.6 mV

Fig. 3.18 : Multifocal run lead II signal

11. R ON T WAVE

Table 3.11 : R on T Wave data

220	200	201	205	200	202	201	202
218	200	201	204	200	201	201	202
217	200	200	202	201	200	200	201
215	201	200	202	201	199	200	201
213	200	200	201	202	199	200	201
211	200	201	200	202	199	200	201
209	200	201	200	201	199	200	202
207	200	200	201	202	199	200	201
206	200	201	200	202	199	200	202
204	200	201	200	203	199	200	201
203	200	201	200	202	199	200	201
202	200	201	200	203	199	200	202
201	201	200	200	202	199	200	202
201	200	201	200	203	198	200	203
200	200	201	201	204	199	200	204
200	200	201	200	204	198	200	204
200	200	201	198	204	198	200	205
200	200	202	195	206	199	200	206
199	200	202	192	206	199	200	206
200	200	201	192	207	199	201	205
199	200	201	197	208	199	200	205
199	200	201	209	209	199	200	205
199	200	201	230	210	199	200	206
199	200	201	253	211	200	200	207
199	200	202	273	212	200	201	207
199	200	201	290	214	200	201	208
199	200	201	290	214	200	200	207
200	200	202	248	215	199	200	208
199	201	201	216	216	200	201	207
199	201	201	198	218	199	200	206
199	200	201	184	218	199	201	205
199	200	202	183	219	200	200	203
199	200	202	190	219	200	200	202
200	200	204	194	219	200	200	202
199	200	204	195	220	200	200	202
199	200	204	197	220	200	201	201
200	200	205	198	219	200	201	200
199	201	206	199	218	200	201	201
199	200	204	198	217	200	201	201
199	200	204	199	215	200	201	200
199	200	204	200	214	200	201	201
200	200	206	199	211	200	201	200
199	201	207	200	210	200	201	200
200	201	206	200	208	201	201	201
200	200	206	199	206	200	201	200
200	200	207	200	205	201	201	199
200	201	208	200	203	200	201	196
200	201	206	200	203	201	201	192

192	180	200	200	199	200	198	199
196	212	200	200	199	200	198	199
207	213	200	200	199	200	198	199
226	214	201	200	199	200	198	199
250	216	200	201	199	200	198	199
270	216	200	200	199	200	198	199
287	218	200	200	199	200	198	199
292	218	200	200	198	200	198	199
254	218	200	200	194	200	198	199
220	219	201	200	191	200	198	199
200	220	200	200	191	200	198	199
202	221	201	200	196	200	199	199
204	221	200	200	200	200	198	199
206	220	200	201	200	200	198	199
208	218	200	200	200	200	198	199
210	217	200	201	200	200	199	199
210	216	201	200	200	200	199	199
210	214	201	201	200	200	198	199
208	212	201	200	200	200	199	199
206	210	201	200	200	200	199	199
204	209	201	200	200	200	199	199
202	207	202	201	200	200	199	199
200	205	200	200	200	200	199	200
199	204	200	200	200	200	199	199
198	202	200	200	200	200	199	199
195	202	200	200	200	200	200	199
192	200	200	201	200	200	199	199
190	201	200	201	200	200	199	199
185	200	199	201	200	200	199	199
185	200	200	201	200	200	199	199
180	200	201	202	200	200	199	198
180	199	200	202	200	200	199	199
175	199	200	202	200	200	199	199
170	200	200	202	200	200	199	199
165	199	200	202	200	201	198	200
160	199	200	202	200	200	199	200
160	200	200	202	200	199	199	201
155	199	200	202	200	199	199	201
150	199	200	202	200	198	199	201
145	200	200	202	200	198	198	203
140	200	201	206	200	198	199	203
135	199	201	207	200	198	199	203
130	199	201	206	200	198	199	202
125	199	200	205	200	198	199	203
125	199	200	203	200	198	199	203
130	200	200	202	200	197	198	204
135	199	201	201	200	198	199	204
140	199	201	200	200	198	199	204
150	199	200	200	200	198	199	204
155	199	201	200	200	198	198	204
160	199	200	200	200	198	198	203
170	199	200	199	200	198	199	202

200	197	192	192	197	192	193	196
199	197	192	192	198	191	193	196
198	197	192	192	196	191	193	197
198	198	192	192	196	191	192	197
198	198	192	193	196	193	193	197
197	198	192	192	194	193	193	196
197	199	192	192	192	193	193	197
197	198	191	191	192	194	193	197
198	199	192	191	191	193	194	197
197	200	192	191	191	194	193	197
197	200	192	191	190	194	193	196
197	200	192	191	191	194	193	197
197	202	192	191	190	194	194	197
197	202	192	192	190	195	193	197
196	202	192	192	190	196	193	197
191	204	192	190	190	196	193	198
189	204	192	192	190	196	194	198
188	206	193	190	190	197	194	198
192	207	193	189	189	198	194	198
202	208	192	191	189	199	194	198
222	208	192	193	190	200	194	198
246	210	193	192	187	201	194	198
265	211	193	191	183	202	194	198
283	212	192	190	182	203	194	198
288	212	193	191	185	204	193	198
250	214	192	192	198	206	194	198
216	214	193	194	216	206	195	199
196	214	192	194	240	207	196	198
182	214	192	194	260	209	194	199
179	214	192	193	278	210	196	199
184	214	192	192	282	210	196	199
190	213	192	192	243	212	196	199
191	211	193	193	209	212	196	199
193	210	192	192	189	213	196	199
194	207	193	193	174	213	197	199
194	206	192	193	172	213	197	199
195	203	192	193	178	212	197	200
195	202	192	192	182	211	197	200
195	200	192	193	184	211	197	200
195	198	193	193	186	208	198	200
195	197	192	193	188	206	197	200
195	196	193	194	190	205	197	200
196	195	192	195	190	202	197	202
196	194	193	195	190	200	196	203
197	194	192	195	189	199	197	204
196	193	192	196	189	198	196	203
196	193	192	197	190	196	196	204
196	192	192	196	190	195	196	206
196	192	192	196	191	195	196	206
196	192	192	197	192	194	196	205
197	192	193	197	192	193	196	206
197	192	192	197	193	194	196	207

207	202	197	206	218	207	202
207	203	200	206	219	206	202
208	201	200	206	220	205	202
208	197	201	206	221	205	202
208	194	202	207	222	204	202
208	194	202	207	222	204	202
207	197	202	208	224	204	202
205	209	203	207	224	203	202
204	229	203	208	224	203	202
203	253	203	209	224	203	203
202	273	203	209	224	203	202
203	290	204	210	223	203	202
202	294	203	211	221	203	202
201	255	203	211	220	203	202
201	223	204	212	218	202	203
200	203	204	212	215	203	203
201	187	204	214	214	202	203
202	183	205	214	211	202	end
202	190	205	216	209	202	
202	195	205	217	208	202	

Refer figure 3.19 R on T wave lead II signal and table 3.11 for R on T wave data

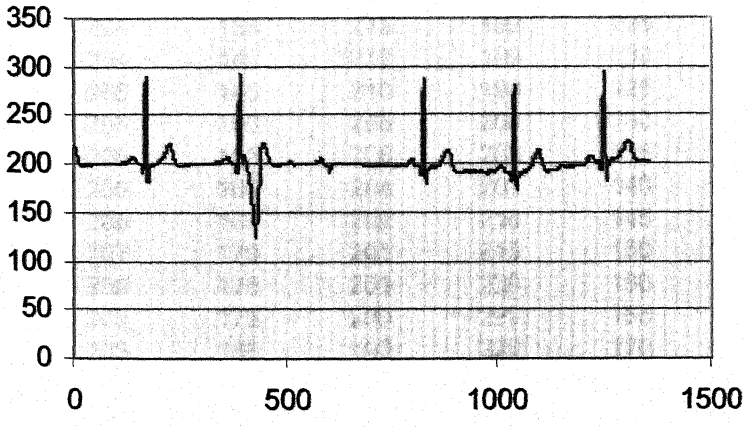


Fig. 3.19 : R on T wave lead II signal

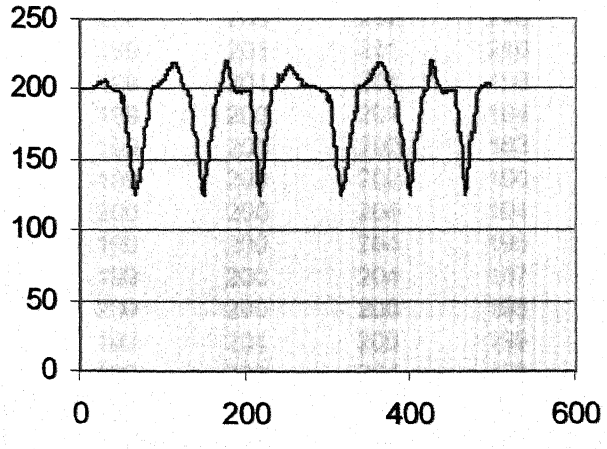
12. VENTRICULAR TACHYCARDIA

Table 3.12 : Ventricular Tachycardia data

200	198	204	145	199	210	200	201
200	194	205	140	199	205	199	201
201	191	206	135	198	206	199	202
200	191	206	130	198	207	199	202
201	196	207	130	199	208	199	202
200	190	208	125	199	209	199	202
201	180	209	130	199	210	199	202
200	180	210	135	199	211	199	203
200	170	211	140	199	212	199	204
200	170	212	145	199	213	198	204
201	160	213	150	199	214	194	205
200	160	214	155	199	215	191	206
200	150	215	160	199	216	191	206
200	150	216	165	199	217	196	207
200	140	216	170	190	216	190	208
201	140	218	170	185	215	180	209
201	130	218	175	180	214	180	210
203	130	218	180	170	213	170	211
203	125	218	185	170	212	170	212
203	130	218	190	165	211	160	213
204	130	218	190	160	210	160	214
205	130	216	195	150	210	150	215
205	135	215	195	140	209	150	216
204	135	213	199	135	208	140	216
204	140	212	199	130	208	140	218
206	145	210	199	125	208	130	218
206	150	208	200	130	208	130	218
206	155	206	205	135	202	125	218
206	160	204	207	140	202	130	218
206	165	202	210	145	202	130	218
207	170	201	215	150	202	130	216
206	175	201	220	160	202	135	215
205	175	200	220	165	202	135	213
203	180	199	220	170	204	140	212
202	180	199	215	170	202	145	210
201	185	195	215	175	202	150	208
200	185	195	210	180	202	155	206
200	201	190	210	185	202	160	204
200	200	190	205	190	202	165	202
200	201	185	205	190	202	170	201
199	201	180	202	200	202	175	201
199	202	175	202	201	202	175	200
199	202	170	202	201	202	180	199
199	202	170	199	201	202	180	199
199	202	165	199	203	201	185	195
199	202	160	199	203	200	185	195
199	203	155	198	203	200	201	190
199	204	150	199	205	200	200	190

185	130	195	210	198	170	170	203
180	135	195	210	199	165	170	203
175	140	199	205	199	160	175	204
170	145	199	205	199	150	180	204
170	150	199	202	199	140	185	204
165	155	200	202	199	135	190	204
160	160	205	202	199	130	190	204
155	165	207	199	199	125	200	203
150	170	210	199	199	130	201	202
145	170	215	199	199	135	201	end
140	175	220	198	199	140	201	
135	180	220	199	190	145	203	
130	185	220	199	185	150	203	
130	190	215	199	180	160	203	
125	190	215	198	170	165	202	

Refer figure 3.20 for Ventricular tachycardia lead II signal for table 3.12 for Ventricular tachycardia data



HR = 150 beats/min
X axis
200= 1200 msec.
Y axis'
200 = 0 V.
280 = 1.6 mV

Fig. 3.20 : Ventricular tachycardia lead II signal

13. HEART BLOCK

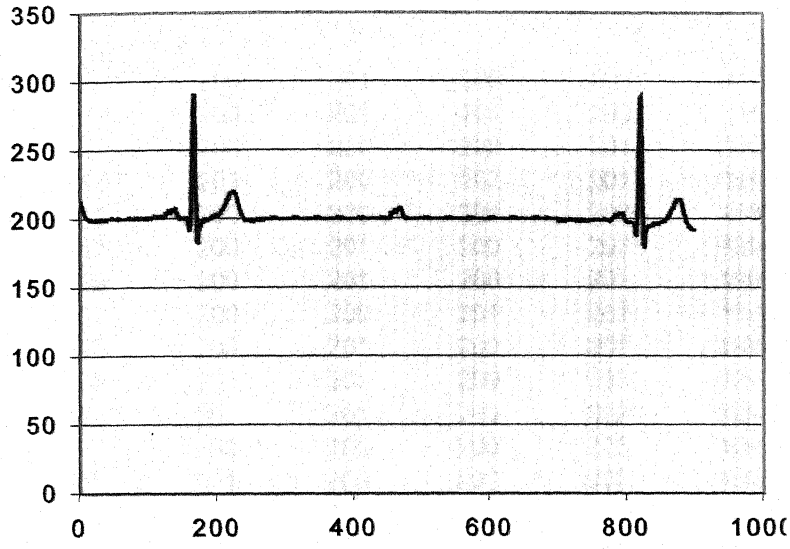
Table 3.13 : Heart Block Data

213	200	200	201	202	199	200	200
211	200	201	200	202	199	200	200
209	200	201	200	201	199	200	200
207	200	200	201	202	199	200	200
206	200	201	200	202	199	200	200
204	200	201	200	203	199	200	200
203	200	201	200	202	199	200	200
202	200	201	200	203	199	200	201
201	201	200	200	202	199	200	200
201	200	201	200	203	198	200	201
200	200	201	201	204	199	200	200
200	200	201	200	204	198	200	201
200	200	201	198	204	198	200	201
200	200	202	195	206	199	200	201
199	200	202	192	206	199	200	200
200	200	201	192	207	199	200	200
199	200	201	197	208	199	200	200
199	200	201	209	209	199	200	200
199	200	201	230	210	199	200	200
199	200	201	253	211	200	200	200
199	200	202	273	212	200	200	200
199	200	201	290	214	200	200	200
199	200	201	290	214	200	200	200
200	200	202	248	215	199	201	200
199	201	201	216	216	200	200	200
199	201	201	198	218	199	201	200
199	200	201	184	218	199	200	200
199	200	202	183	219	200	201	200
199	200	202	190	219	200	201	200
200	200	204	194	219	200	201	200
199	200	204	195	220	200	200	200
199	200	204	197	220	200	200	200
200	200	205	198	219	200	200	200
199	201	206	199	218	200	200	200
199	200	204	198	217	200	200	200
199	200	204	199	215	200	200	200
199	200	204	200	214	200	200	200
200	200	206	199	211	200	200	200
199	201	207	200	210	200	200	200
200	201	206	200	208	201	200	201
200	200	206	199	206	200	200	200
200	200	207	200	205	201	200	201
200	201	208	200	203	200	200	200
200	201	206	200	203	201	200	201
200	201	205	200	202	201	200	201
200	201	204	200	201	201	200	201
200	200	202	201	200	200	200	200
201	200	202	201	199	200	200	200

200	200	200	201	200	200	200	199
200	200	200	201	200	200	200	199
200	200	200	200	200	200	200	199
200	201	200	200	200	200	200	199
200	200	200	200	200	200	198	199
200	201	200	200	200	200	198	199
200	200	200	200	200	200	198	199
200	201	200	200	201	200	199	199
200	201	200	200	200	200	198	199
200	201	200	200	201	200	198	199
200	200	200	200	200	200	198	199
200	200	200	200	201	200	199	199
200	200	200	200	201	200	199	199
200	200	200	200	201	200	198	199
200	200	200	200	200	200	199	199
200	200	201	200	200	200	199	199
200	200	200	200	200	200	199	199
200	202	201	200	200	200	199	199
200	204	200	200	200	200	199	200
200	204	201	200	200	201	199	199
200	204	201	200	200	200	199	199
200	205	201	200	200	201	200	199
200	206	200	200	200	200	199	199
201	204	200	200	200	201	199	199
200	204	200	200	200	201	199	199
201	204	200	200	200	201	199	199
200	206	200	200	200	200	199	198
201	207	200	201	200	200	199	199
201	206	200	200	200	200	199	199
201	206	200	201	200	200	199	199
200	207	200	200	200	200	198	200
200	208	200	201	200	200	199	200
200	206	200	201	200	200	199	201
200	205	200	201	200	200	199	201
200	204	200	200	200	200	199	201
200	202	200	200	200	200	198	203
200	202	200	200	200	200	199	203
200	201	200	200	200	200	199	203
200	200	200	200	200	200	199	202
200	200	200	200	201	201	199	203
200	201	200	200	200	200	199	203
200	200	200	200	201	200	198	204
200	200	200	200	200	200	199	204
200	200	200	200	201	200	199	204
200	200	200	200	201	200	199	204
200	200	200	200	201	200	198	204
200	200	200	200	200	200	198	203
200	200	201	200	200	200	199	202
200	200	200	200	200	200	199	200
200	200	201	200	200	200	199	199
200	200	200	200	200	200	199	198
200	200	201	200	200	200	199	198

198	198	194	196	205	203	206
197	198	193	196	206	203	205
197	199	194	196	207	204	205
197	198	193	196	207	203	204
198	199	193	196	207	203	204
197	200	193	197	208	204	204
197	200	192	197	208	204	203
197	200	193	197	208	204	203
197	202	193	196	208	205	203
197	202	193	197	207	205	203
196	202	193	197	205	205	203
191	204	194	197	204	206	203
189	204	193	197	203	206	203
188	206	193	196	202	206	202
192	207	193	197	203	206	203
202	208	194	197	202	207	202
222	208	193	197	201	207	202
246	210	193	197	201	208	202
265	211	193	198	200	207	202
283	212	194	198	201	208	202
288	212	194	198	202	209	202
250	214	194	198	202	209	202
216	214	194	198	202	210	202
196	214	194	198	202	211	202
182	214	194	198	203	211	202
179	214	194	198	201	212	202
184	214	194	198	197	212	202
190	213	193	198	194	214	202
191	211	194	198	194	214	203
193	210	195	199	197	216	202
194	207	196	198	209	217	202
194	206	194	199	229	218	202
195	203	196	199	253	219	202
195	202	196	199	273	220	203
195	200	196	199	290	221	203
195	198	196	199	294	222	203
195	197	196	199	255	222	
195	196	197	199	223	224	
196	195	197	199	203	224	
196	194	197	200	187	224	
197	194	197	200	183	224	
196	193	197	200	190	224	
196	193	198	200	195	223	
196	192	197	200	197	221	
196	192	197	200	200	220	
196	192	197	202	200	218	
197	192	196	203	201	215	
197	199	197	204	202	214	
197	198	196	203	202	211	
197	196	196	204	202	209	
197	195	196	206	203	208	
198	195	196	206	203	207	

Refer figure 3.21 for Heart Block lead II signal and table 3. 13 for Heart Block data

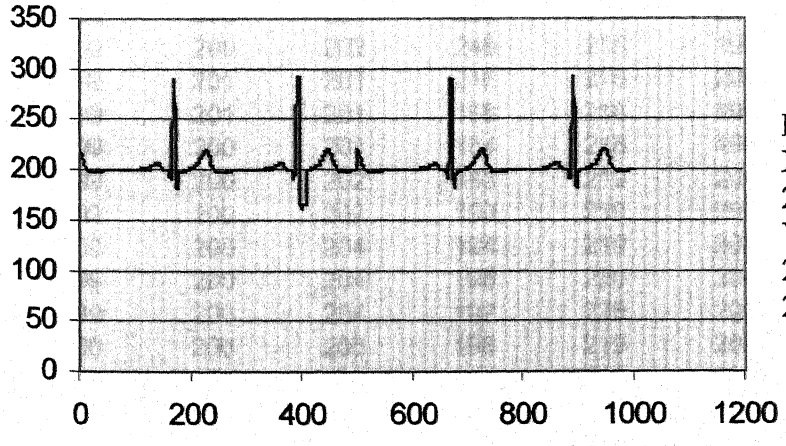


HR = 46 beats/min
X axis
200= 400 msec.
Y axis'
200 = 0 V.
280 = 1.6 mV

Fig. 3.21 : Heart Block lead II signal

14. VPB1 (VENTRICULAR PREMATURE BEAT 1)

Refer figure 3.22 for VPB 1 lead II signal and table 3.14 for VPB 1 lead data



HR = 75 beats/min
X axis
200= 800 msec.
Y axis'
200 = 0 V.
280 = 1.6 mV

Fig. 3.22 : VPB 1 lead II signal

Table 3.14 : VPB 1 (Ventricular Premature Beat 1) data

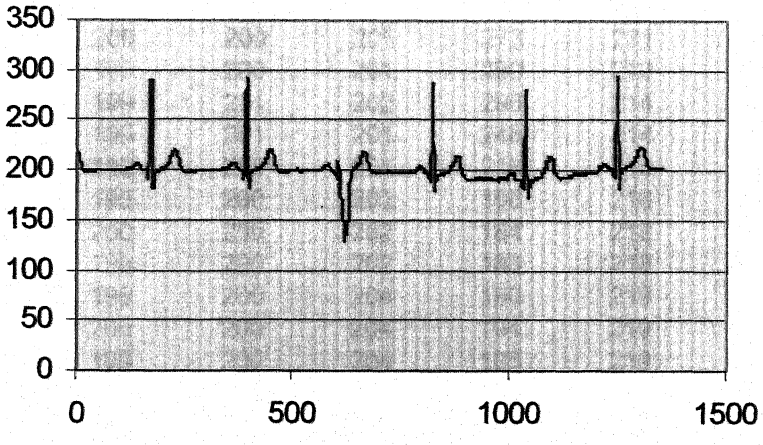
220	200	201	205	200	202	201	202
218	200	201	204	200	201	201	202
217	200	200	202	201	200	200	201
215	201	200	202	201	199	200	201
213	200	200	201	202	199	200	201
211	200	201	200	202	199	200	201
209	200	201	200	201	199	200	202
207	200	200	201	202	199	200	201
206	200	201	200	202	199	200	202
204	200	201	200	203	199	200	201
203	200	201	200	202	199	200	201
202	200	201	200	203	199	200	202
201	201	200	200	202	199	200	202
201	200	201	200	203	198	200	203
200	200	201	201	204	199	200	204
200	200	201	200	204	198	200	204
200	200	201	198	204	198	200	205
200	200	202	195	206	199	200	206
199	200	202	192	206	199	200	206
200	200	201	192	207	199	201	205
199	200	201	197	208	199	200	205
199	200	201	209	209	199	200	205
199	200	201	230	210	199	200	206
199	200	201	253	211	200	200	207
199	200	202	273	212	200	201	207
199	200	201	290	214	200	201	208
199	200	201	290	214	200	200	207
200	200	202	248	215	199	200	208
199	201	201	216	216	200	201	207
199	201	201	198	218	199	200	206
199	200	201	184	218	199	201	205
199	200	202	183	219	200	200	203
199	200	202	190	219	200	200	202
200	200	204	194	219	200	200	202
199	200	204	195	220	200	200	202
199	200	204	197	220	200	201	201
200	200	205	198	219	200	201	200
199	201	206	199	218	200	201	201
199	200	204	198	217	200	201	201
199	200	204	199	215	200	201	200
199	200	204	200	214	200	201	201
200	200	206	199	211	200	201	200
199	201	207	200	210	200	201	200
200	201	206	200	208	201	201	201
200	200	206	199	206	200	201	200
200	200	207	200	205	201	201	199
200	201	208	200	203	200	201	196
200	201	206	200	203	201	201	192

192	212	200	199	200	205	202	199
196	212	200	200	200	204	202	199
207	213	200	199	201	202	201	199
226	214	201	200	201	202	202	199
250	216	200	200	201	201	202	199
270	216	200	200	201	200	203	198
270	218	200	200	200	200	202	199
292	218	200	200	200	201	203	198
292	218	200	200	200	200	202	198
293	219	201	200	201	200	203	199
292	220	200	200	201	200	204	199
292	221	201	201	200	200	204	199
173	221	220	200	201	200	204	199
162	220	218	200	201	200	206	199
165	218	217	200	201	201	206	199
165	217	215	200	201	200	207	200
165	216	213	200	200	198	208	200
165	214	211	200	201	195	209	200
165	212	209	200	201	192	210	200
165	210	207	200	201	192	211	199
165	209	206	201	201	197	212	200
165	207	204	200	202	209	214	199
165	205	203	200	202	230	214	199
165	204	202	200	201	253	215	200
201	202	201	200	201	273	216	200
201	202	201	200	201	290	218	200
201	200	200	200	201	290	218	200
201	201	200	200	201	248	219	200
201	200	200	200	202	216	219	200
201	200	200	200	201	198	219	200
201	200	199	200	201	184	220	200
201	199	200	200	202	183	220	200
202	199	199	200	201	190	219	200
201	200	199	200	201	194	218	200
202	199	199	200	201	195	217	200
203	199	199	200	202	197	215	201
202	200	199	201	202	198	214	200
203	199	199	201	204	199	211	201
203	199	199	200	204	198	210	200
204	200	200	200	204	199	208	201
204	200	199	200	205	200	206	201
205	199	199	200	206	199	205	201
204	199	199	200	204	200	203	200
204	199	199	200	204	200	203	200
205	199	199	200	204	199	202	200
206	200	200	201	206	200	201	200
206	199	199	200	207	200	200	200
207	199	199	200	206	200	199	200
208	199	200	200	206	200	199	200
208	199	199	200	207	200	199	200
209	199	199	201	208	201	199	200
210	199	199	201	206	201	199	200

200	201	204	200	197	205	218	200
200	201	204	201	199	204	217	200
200	201	205	200	200	204	216	199
200	201	206	200	199	205	214	199
200	201	206	201	200	206	212	199
200	201	205	200	200	206	210	199
200	201	205	199	200	207	209	200
201	201	205	196	200	208	207	199
200	201	206	192	201	208	205	199
200	201	207	192	201	209	204	199
200	201	207	196	201	210	202	199
200	202	208	207	201	212	202	199
201	202	207	226	201	212	200	199
201	201	208	250	201	213	201	200
200	201	207	270	201	214	200	200
200	201	206	287	201	216	200	200
201	201	205	292	202	216	200	201
200	202	203	254	201	218	199	200
201	201	202	220	202	218	199	200
200	202	202	200	203	218	200	200
200	201	202	186	202	219	199	200
200	201	201	183	203	220	199	200
200	202	200	190	203	221	200	201
201	202	201	195	204	221	199	200
201	203	201	196	204	220	199	201

15. VPB 2 (VENTRICULAR PREMATURE BEAT 2)

Refer figure 3.23 for VPB 2 lead II signal and table 3.15 for VPB 2 lead data



HR = 68 beats/min
X axis
500= 2 sec.
Y axis
200 = 0 V.
280 = 1.6 mV

Fig. 3.23 : VPB 2 lead II signal

Table 3.15 : VPB2 (Ventricular Premature Beat2) data

220	200	201	207	199	208	200	201
218	200	201	208	200	206	201	201
217	200	201	206	200	205	200	201
215	200	201	205	200	203	201	201
213	201	200	204	200	203	200	201
211	200	200	202	200	202	201	201
209	200	200	202	201	201	201	201
207	200	201	201	201	200	201	202
206	200	201	200	202	199	200	202
204	200	200	200	202	199	200	201
203	200	201	201	201	199	200	201
202	200	201	200	202	199	200	201
201	200	201	200	202	199	200	201
201	201	201	200	203	199	200	202
200	200	200	200	202	199	200	201
200	200	201	200	203	199	200	202
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199	200	201	200	204	198	200	202
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199	200	201	192	206	198	200	204
199	200	201	192	206	199	200	204
199	200	201	197	207	199	200	205
199	200	201	209	208	199	200	206
199	200	201	230	209	199	201	206
199	200	202	253	210	199	200	205
200	200	201	273	211	199	200	205
199	200	201	290	212	200	200	205
199	201	202	290	214	200	200	206
199	201	201	248	214	200	201	207
199	200	201	216	215	200	201	207
199	200	201	198	216	199	200	208
200	200	202	184	218	200	200	207
199	200	202	183	218	199	201	208
199	200	204	190	219	199	200	207
200	200	204	194	219	200	201	206
199	200	204	195	219	200	200	205
199	201	205	197	220	200	200	203
199	200	206	198	220	200	200	202
199	200	204	199	219	200	200	202
200	200	204	198	218	200	201	202
199	200	204	199	217	200	201	201
200	201	206	200	215	200	201	200
200	201	207	199	214	200	201	201
200	200	206	200	211	200	201	201
200	200	206	200	210	200	201	200

201	205	199	200	203	201	198	199
200	206	200	200	202	200	197	198
200	206	199	201	201	201	198	199
201	207	199	201	200	201	198	199
200	208	199	200	200	202	198	199
199	208	199	201	200	202	198	198
196	209	199	200	200	202	198	198
192	210	199	200	199	202	198	199
192	212	200	200	199	202	198	199
196	212	200	200	199	203	198	199
207	213	200	200	199	204	198	199
226	214	201	200	199	204	198	199
250	216	200	201	199	205	198	199
270	216	200	200	199	206	198	199
287	218	200	200	199	206	198	199
292	218	200	200	198	207	198	199
254	218	200	200	194	208	198	199
220	219	201	200	191	209	198	199
200	220	200	200	191	210	198	199
186	221	201	200	196	211	199	199
183	221	200	200	209	212	198	199
190	220	200	201	200	213	198	199
195	218	200	200	190	214	198	199
196	217	200	201	190	215	199	199
197	216	201	200	185	216	199	199
199	214	201	201	185	216	198	199
200	212	201	200	180	218	199	199
199	210	201	200	170	218	199	199
200	209	201	200	160	218	199	199
200	207	202	201	160	218	199	199
200	205	200	200	160	218	199	200
200	204	200	200	150	218	199	199
201	202	200	200	150	216	199	199
201	202	200	200	140	215	200	199
201	200	200	201	130	213	199	199
201	201	200	201	130	212	199	199
201	200	199	203	135	210	199	199
201	200	200	203	135	208	199	199
201	200	201	203	135	206	199	198
201	199	200	204	140	204	199	199
202	199	200	205	140	202	199	199
201	200	200	205	150	201	199	199
202	199	200	204	150	201	198	200
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202	200	200	206	165	199	199	201
203	199	200	206	165	199	199	201
203	199	200	206	170	198	199	201
204	200	200	206	200	198	198	203
204	200	201	206	200	198	199	203
205	199	201	207	201	198	199	203
204	199	201	206	200	198	199	202
204	199	200	205	200	198	199	203

203	197	194	192	195	189	199	197
204	196	193	192	196	189	198	196
204	196	193	192	197	190	196	196
204	196	192	192	196	190	195	196
204	196	192	192	196	191	195	196
204	196	192	192	197	192	194	196
203	197	192	193	197	192	193	196
202	197	192	192	197	193	194	196
200	197	192	192	197	192	193	196
199	197	192	192	198	191	193	196
198	197	192	192	196	191	193	197
198	198	192	192	196	191	192	197
198	198	192	193	196	193	193	197
197	198	192	192	194	193	193	196
197	199	192	192	192	193	193	197
197	198	191	191	192	194	193	197
198	199	192	191	191	193	194	197
197	200	192	191	191	194	193	197
197	200	192	191	190	194	193	196
197	200	192	191	191	194	193	197
197	202	192	191	190	194	194	197
197	202	192	192	190	195	193	197
196	202	192	192	190	196	193	197
191	204	192	190	190	196	193	198
189	204	192	192	190	196	194	198
188	206	193	190	190	197	194	198
192	207	193	189	189	198	194	198
202	208	192	191	189	199	194	198
222	208	192	193	190	200	194	198
246	210	193	192	187	201	194	198
265	211	193	191	183	202	194	198
283	212	192	190	182	203	194	198
288	212	193	191	185	204	193	198
250	214	192	192	198	206	194	198
216	214	193	194	216	206	195	199
196	214	192	194	240	207	196	198
182	214	192	194	260	209	194	199
179	214	192	193	278	210	196	199
184	214	192	192	282	210	196	199
190	213	192	192	243	212	196	199
191	211	193	193	209	212	196	199
193	210	192	192	189	213	196	199
194	207	193	193	174	213	197	199
194	206	192	193	172	213	197	199
195	203	192	193	178	212	197	200
195	202	192	192	182	211	197	200
195	200	192	193	184	211	197	200
195	198	193	193	186	208	198	200
195	197	192	193	188	206	197	200
195	196	193	194	190	205	197	200
196	195	192	195	190	202	197	202
196	194	193	195	190	200	196	203

204	203	273	203	208	224	204	202
203	202	290	203	209	224	203	202
204	201	294	204	209	224	203	202
206	201	255	203	210	224	203	202
206	200	223	203	211	223	203	203
205	201	203	204	211	221	203	202
206	202	187	204	212	220	203	202
207	202	183	204	212	218	203	202
207	202	190	205	214	215	202	202
207	202	195	205	214	214	203	203
208	203	197	205	216	211	202	203
208	201	200	206	217	209	202	203
208	197	200	206	218	208	202	end
208	194	201	206	219	207	202	
207	194	202	206	220	206	202	
205	197	202	207	221	205	202	
204	209	202	207	222	205	202	
203	229	203	208	222	204	202	
202	253	203	207	224	204	202	

3.4 CONCLUSION

Using the eintheven's laws and data of normal lead II signals obtained at everyone msec. The other lead II signals of different arrhythmias are simulated and when plotted on Excel the simulated lead II signals resemble the morphology of arrhythmia signals. Using this data not only above 17 arrhythmias but other life threatening arrhythmia can be simulated. Thus this data base is useful for simulating any arrhythmia lead II signal and hence is an effective tool for teaching arrhythmias.

CHAPTER – IV

ECG/ARRHYTHMIA SIMULATOR USING MICROCONTROLLER

4.1 INTRODUCTION

Microcontroller 8752 is a 8 bit microcontroller with 8 k EPROM, two timer counters and four 8 bits ports. Because of this features, it is used for ECG arrhythmias simulation so as a portable device can be developed and is useful in carrying at any CCU for checking ECG/arrhythmia monitor performance. The designing and programming algorithms are explained in the following sessions. Figure 4.1 shows block diagram of ECG/arrhythmia simulator using microcontroller.

Microcontroller 8752 [97, 98] is used to control all the operations of the instrument like detection of the keypress, get the keycode, branch to the corresponding 9 arrhythmia waveforms. It controls the synchronous operation of all the circuits. Refer figure 16 for Block Diagram of ECG/Arrhythmia Simulator using Microcontroller.

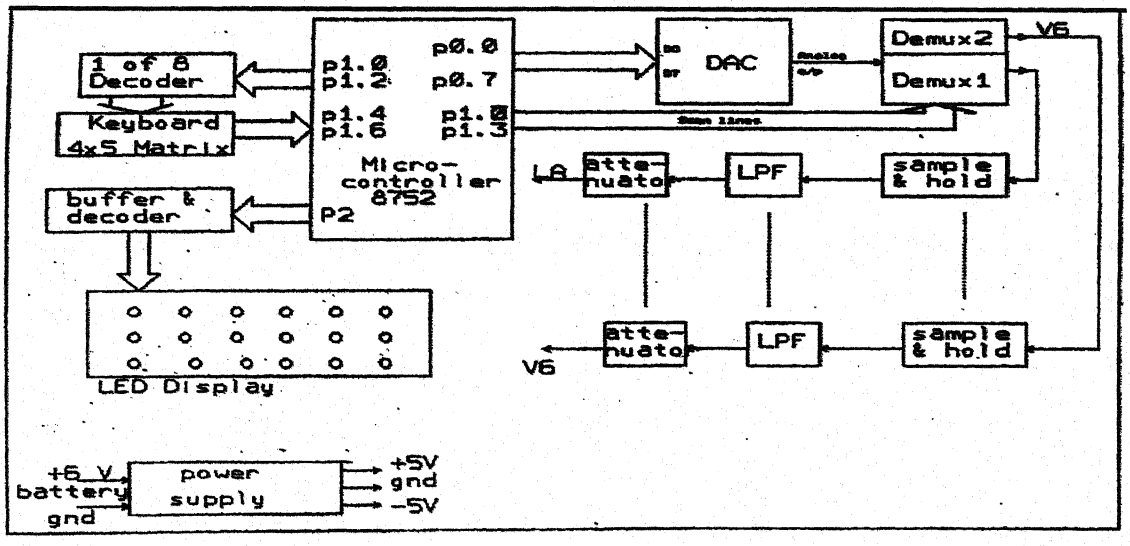

Fig. 4.1 : Block Diagram of ECG/Arrhythmia Simulator using Microcontroller.

The digital data stored in the memory for any arrhythmia waveform detected is passed to the digital to analog converter which converts the signal in a bipolar range.

An analog demultiplexer which is selected using four bits of microcontroller port is used to generate nine waveforms. This is essential to output all nine lead signals at a time with the restriction of being able to output only one digital data on the microcontroller port at a time.

The demultiplexer provides analog signal which is held by sample and hold circuit till the next data appears on it. This held signal is then passed through second order low pass filter with gain of 40 dB/decade.

The waveforms are generated at amplitude levels of 1V, 1.5V, or 2V and hence an attenuator of 1:1000 is used to provide signals in the mV range.

Keyboard is scanned at the rate at which the 9 circuits are enabled. The keyboard matrix is formed using 1 of 8 decoder and 3 port lines of the microcontroller. In all 20 keys are used.

Display is provided to indicate the arrhythmia selected. On any keypress the corresponding LED glows indicating the user the operation or arrhythmia selected. The display consists of 18 LEDs corresponding to the 18 arrhythmias selected using 1 of 16 decoder and 1 of 8 decoder. The LEDs are connected after the buffer to raise the current capability of the signal coming from the microcontroller.

Power supply is used to generate +5V/-5V for the analog and the digital Ics used in the circuit. Depending on the current requirement of each IC the total current being consumed by the circuit is calculated. The power supply is a DC to DC converter.

To make the instrument portable and to avoid the 50Hz noise signal a 6V lead acid battery (1.2Ah) is used. Using a switching regulator and appropriate transformer the supply is generated and supplied all over the circuit.

4.2 DESIGN

The development work consists of hardware designing and software development for the same. The hardware consists of MICROCONTROLLER as the unit which controls time defined specific functions. It is used for,

i) Detection of key pressed and debouncing of the keys.

ii) Recognize the selected signal and

iii) Accordingly branch to respective memory location containing the digitized waveform.

The Software consists of

i) Fetching data for the signals.

ii) Storing the data in proper sequence in EPROM,

iii) Programming the MICROCONTROLLER to properly execute the function of key detection and

iv) Proper sequential output of the data to DAC to generate the required waveform.

For Normal ECG and 17 Arrhythmias, the data for RA, LA, LL, V1, V2, V3, V4, V5 and V6 are stored in EPROM of 8752H (8K) in digitized fashion. This digital data on selection through keyboard is outputted to digital to analog converter (DAC) and corresponding LED is glow to indicate which arrhythmia is selected. The analog output from DAC is given to input of analog Demultiplexer, which is scanned at same rate as keyboard is scanned and the analog output from DEMUX is given to sample and hold circuit, as samples are outputted at 222 microseconds for each waveform and this data is to be held for 2 miliseconds. After the sample and hold circuit, low pass filter is used to smoothen the waveform and to eliminate the high frequency as well as power supply interference. The output obtained is 1V, 1.5V, or 2 V. Hence it is attenuated with 1 : 1000 voltage divider circuit to obtain ECG of 1mV, 1.5mV, and 2mV signal.

4.2.1 DESIGNING OF POWER SUPPLY

Refer figure 4.2 for Power Supply Circuit for ECG/Arrhythmia Simulator using Microcontroller.

To avoid the 50Hz noise from mains, the Instrument is battery operated. So the power supply is to be generated from the battery. A single battery is used to generate DUAL POWER supplies since the DEMUX and operational amplifiers used are operated at +5V. So +6V battery is used to generate +5V and -5V. The basic principal used here is DC to AC and again AC to DC conversion.

DC to AC is converted using inbuit oscillator of 40K Hz from IC LT 1070 [99] which is a switching regulator.

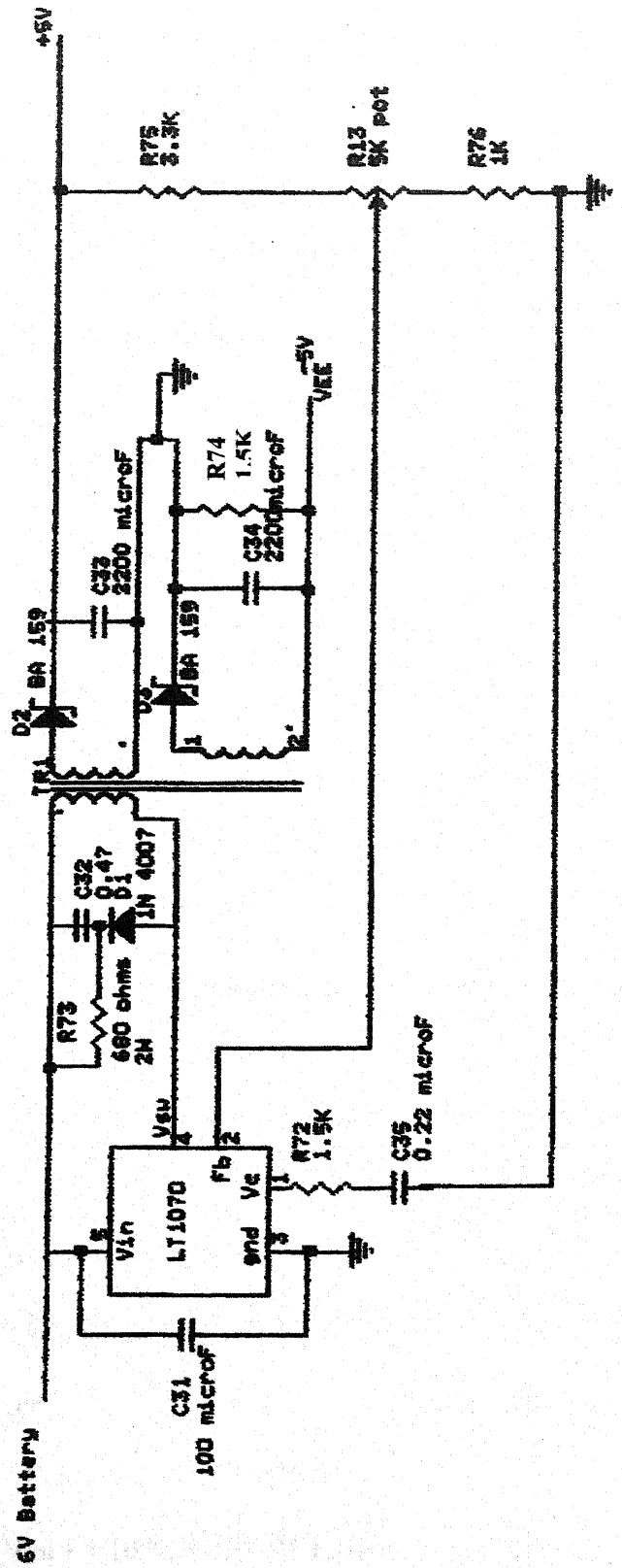

Fig. 4.2 : Power Supply Circuit for ECG/Arrhythmia Simulator using Microcontroller.

Features of LT1070

1) Wide input voltage range from 3V to 60V.

2) Low quiescent current of 6mA.

3) Internal 1.25 A Switch.

4) Very few external parts required.

5) Self protected against overloads.

6) Operates in nearly all switching topologies.

7) Shutdown mode draws only 50 micro AMPS supply current.

8) Flyback regulated mode has fully floating outputs.

9) Can be externally synchronize.

Lprimary

Output Power is : (+5V * 0.4A) + (5V * 0.1A) = 2.5 WATT

I max = 4 * Pout/#* VIN min

where # = efficiency = 0.7 = Vout/Vin

hence I max = 2.8 AMPS

As L di/dt = V where di = Imax and dt = 50% of T = 0.5T also T = 1/f where f is the oscillation frequency of LT 1070

Hence L = 22.3 micro H

As primary : secondary = 1:2

Therefore Lprimary = 22.3 micro H and

Total Lsecondary = 44.6 mciro H

Output divider

R75 = Vout - Vref/Vref * R76

Vref = 1.244V of LT 1070, R76=1.24 K ohms

hence R75 = 3.74 K ohms

Snubber design

$$Rsnb = \frac{2* [Vr^2-Vr * (Vout/N)]}{(Iprimary) ^ 2 * (Li)*f}$$

where Vr = voltage across Snubber resistor

hence Rsnb = 855.67 OHMS i.e. 860 ohms standard value

Pr = Vr^ 2 /R let Vr = 30V

Therefore Pr = 1 watt, providing safety factor of 2

Pr = 2 watts & Rsnb i.e. R73 = 860 ohms/ 2 watt

C3 is not critical but should be large enough and can be calculated as C3 = Vr / R*f*Vs

where Vs is voltage ripple across C 3 = 1.85V

hence C3 as 0.471 micro F

Output diodes D2 and D3

$$Id (peak) = I out * \frac{(1+ Vout + Vf)}{(N * Vin)}$$

$$= 0.4* [1 + ((5+0.7)/6)] = 0.78 amps$$

Power dissipation is given by

Vf * Iout = 0.7 * 0.4 = 0.28 watt

hence diode selected is BA159 which is a schottky diode.

Output peak to peak ripple voltage is given by

$$Vpp = \frac{Iout}{f*C* (1+N*Vin/Vout)} + ESR * (Iout) * (1+Vout/N*Vin)$$

where ESR = is the effective series resistance of C 1 = 0.02 ohms

Now Vpp = 20 mV so C can be calculated as,

$$20\ mV = \frac{0.4}{40*10\ ^\wedge\ 3*C\ (1+6/5)} + (0.02 * 0.4) * (1+5/6)$$

Therefore C = 2160 micro F

Selecting standard value C33 = C34 = 2200 micro F /16 V

Ripple factor = Vac / Vdc = 20mV/5V = 0.04

Battery specifications

Sealed Lead acid battery, type : ES 1.2 -6

Normal Voltage 6V

Rated capacity (AH) : 1.0 AH.

Maximum charging current : 0.3A

Charging voltage : Stand by use - 6.75 V to 6.9 V

Cycle use - 7.2 V to 7.5 V

Dimensions : L = 97 mm

W = 25 mm

H = 50 mm

Weight : 0.3 Kg.

4.2.2 DESIGNING OF DIGITAL SECTION

Refer figure 4.3 for Circuit Diagram of ECG/Arrhythmia Simulator using microcontroller – Digial Section

Microcontroller 8752

The microcontroller 8752H controlling all the operations has the following features:

i) 8 bit CPU

ii) On chip oscillator

iii) 8k bytes of EPROM

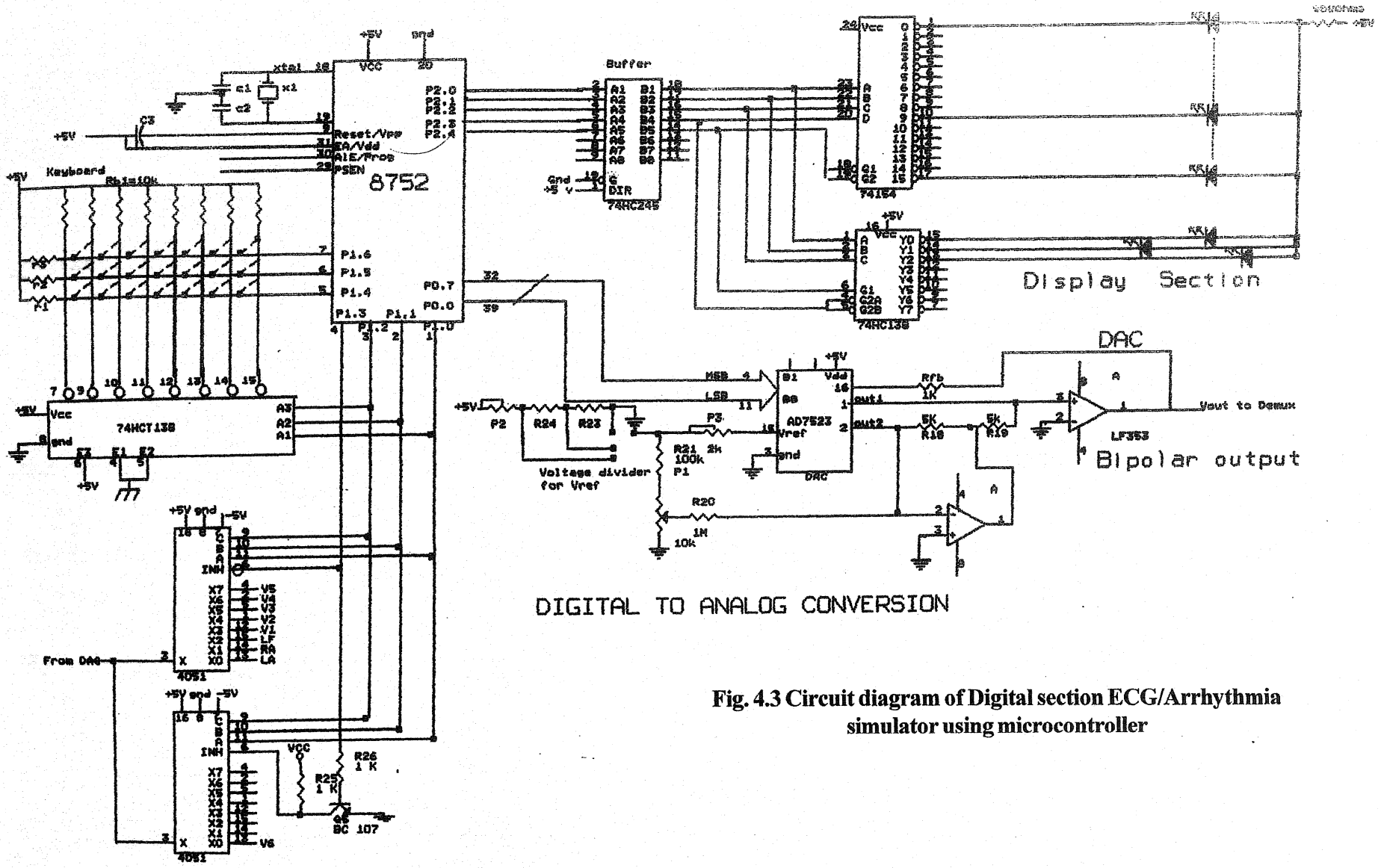

Fig. 4.3 Circuit diagram of Digital section ECG/Arrhythmia simulator using microcontroller

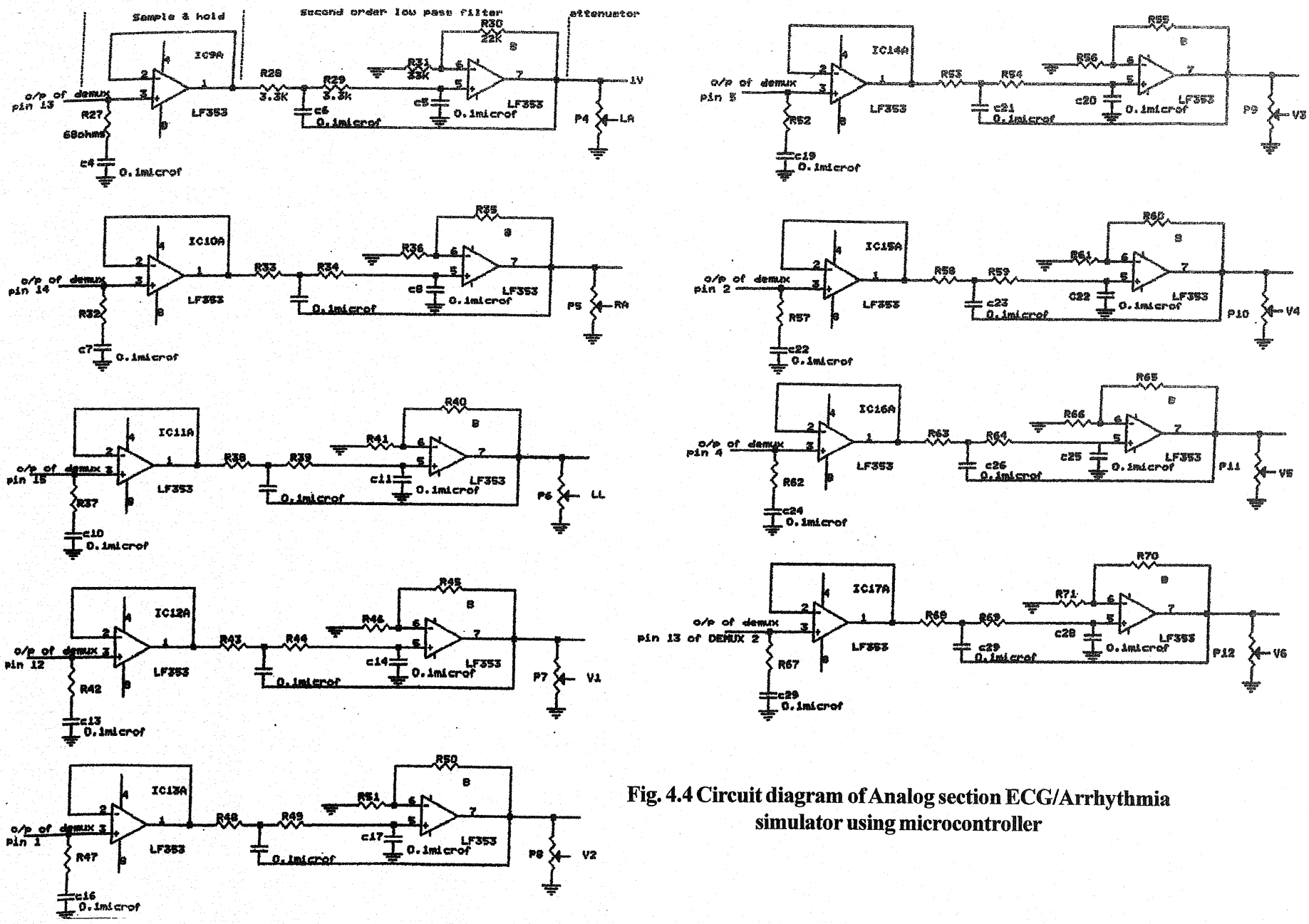

Fig. 4.4 Circuit diagram of Analog section ECG/Arrhythmia simulator using microcontroller

iv) 128 bytes of RAM

v) 21 special function registers

vi) 32 I /O lines

vii) 2, 16 bit timers/counters.

viii) A 5 source interrupt structure with two priority levels.

ix) A full duplex serial port.

x) bit addressability for boolean processing.

Crystal used for the clock generation is 11.05924 MHz. with capacitors 5pF and 10pF prescribed by the data sheets to form the perfect oscillations. At reset pin a capacitor of 10pF /16V is connected along with +5V which is connected to the positive terminal of capacitor, thus avoiding the in between reset of the microcontroller.

EA/VDD pin is connected to Vcc as no external mem
output pins they are kept open.

Port assignments

1) PORT 0 which is 8 bit port is connected to digital to analog converter which is assigned as Output Port.

2) PORT 1 where the lower nibble or lower port is used as scan lines to scan the keyboard. These bits are also used to scan the DEMUX. These are hence assigned as Output Port.

3) PORT 1 where the upper pins i.e. P1.4, P1.5 and P1.6 are used as input pins to read the keyboard status.

4) PORT 2 where the lower 5 pins are used for display section. Since the 18 LEDs are to glow, to avoid 18 port pins, 5 pins are connected to 4 to 16 decoder and 3 to 8 decoder so 24 output lines are available out of which 18 are used.

Digital to analog converter (AD 7523)

As digital data from microcontroller is needed to be converted into analog signal, a digital to analog converter is used. 8 bits data is sufficient to get the proper analog signal and hence 8 bits DAC is used which will have very low settling time. DAC used here is INTERSIL JEET MOSFET amplifiers AD 7523. It's main features are :

1) DTL/TTL/CMOS compatible.

2) +5V to + 15V volts supply range.

3) fast settling time = 100 nanosecs.

4) INTERSIL AD7523 provides accurate four quadrant multiplication, full military temperature range operation, full input protection from damage due to static discharge by clamps to V+ and very low power dissipation make it a very versatile converter.

AD7523 is used in bipolar mode, R18 and R19 must be matched for 0.1% or better. R17 and P3 is used for gain adjustment. R20, R21 and P1 are used to adjust Vout = 0V at input of 10000000

Here 1LSB = 2 ^(- 7)*Vref = (1/128) * Vref.

DIGITAL INPUT MSB	LSB	ANALOG OUTPUT
1111	1111	-Vref * (127/128)
1000	0001	-Vref * (1/128)
1000	0000	0
0111	1111	+Vref * (1/128)
0000	0001	+Vref * (127/128)
0000	0000	+Vref * (128/128)

4.2.3 DESIGNING OF ANALOG SECTION

Refer figure 4.4 for Circuit Diagram of analog section of ECG/Arrhythmia Simulator using Microcontroller.

Analog demultiplexer (74HC4051)

The output from DAC is given to analog demultiplexer [100] which are switches operated on the condition of input select lines It is 3 to 8 demultiplexer.

The Channel select and enable inputs are compatible with standard CMOS outputs with pull up resistors, they are compatible with LSTTL outputs also.

Its features are,

1) fast switching and propagation speeds.

2) low cross talk between switches.

3) Diode protection on all input/outputs.

4) Analog power supply range (VCC-GND) = 2.0 to 6.0 V.

5) Improved linearity and lower ON resistance.

6) Low noise.

Function Table of 74HC4051

CONTROL INPUTS				ON CHANNELS
P1.3	P1.2	P1.1	P1.0	&
EN	C	B	A	OUTPUTS
0	0	0	0	X0=LA ----------|
0	0	0	1	X1=RA |
0	0	1	0	X2=LL |
0	0	1	1	X3=V1 |
0	1	0	0	X4=V2 DEMUX 1
0	1	0	1	X5=V3 |
0	1	1	0	X6=V4 |
0	0	1	1	X7=V5 ----------|
1	0	0	0	X0 = V6 DEMUX 2

Sample and Hold Circuit

The output from demultiplexer channel is given to sample and hold circuit as the output rate for each signal on corresponding channel of DEMUX is 222 microsecs but that data is to be held for 2 msecs at the output, sample and hold circuit is required at each output pin of demultiplexer. Such nine sample and hold circuits are used. OPAMP used is LF353 (dual opamp) [101].

As,

T sample > 5RC

where R and C are the sample and hold components at input of opamp LF 353.

as T sample = 222 microsecs, let C = 0.1 micro F

and R acan be designed as

Output impedance of DAC + On resistance of DEMUX CHANNEL + R

let R = 60 OHMS

Total R = 70 + 250 + 68 = 388 ohms

hence RC = 5 * 388 * 0.1 * 10 ^ -6 = 194 microsecs

This satisfies T sample > 5 RC,

since 222 microsecs > 194 microsecs

Low pass filter

Since the output of sample and hold circuit is the waveform with each byte outputted at the rate of 2 msecs and hence to smoothen the waveform and to avoid high frequency and mains frequency interference a low pass filter (LPF) is designed

for LPF T = 2 * 3.1414 * R * C

i. e. 2 msecs = 2 * 3.1414 * R * C

let C = 0.1 microF hence R = 3.3 K ohms

also A = 3 - @ where @ = 1.414 = damping coefficient

hence A = 3 - 1.414 = 1.586

i.e.1.586 = 1 + (Rf / Ri)

Rf/Ri = 1.586 - 1 =0.586

for minimum dc offset,

Rf // Ri = 2R

(Rf * Ri)/(Rf + Ri) =2R

Ri *(0.586Ri)/1.586*Ri =2 * 3.3k

we get Ri = 34.46K OHMS

selecting nearest standard value, Ri = 33K ohms and Rf=22K ohms

Recalculated A = 1 + (Rf /Ri) =1 + 22/33 = 1. 6666

Attenuator

Since all outputs obtained are of magnitude 2V, 1. 5V and lV they are attenuated by the voltage divider of 1: 1000, to obtain 2 mV, 1.5 mV and 1.0 mV signal. This attenuation is done by using 1OOK potentiometer.

4.2.4. KEYBOARD AND DISPLAY SECTION

This section gives the designing of keyboard and display part used in portable simulator front panel.

Keyboard Section

In all ,20 push-button keys are used in matrix form of 8x3. Two keys are kept for future use and rest of the 18 keys are used to select 17 different arrhythmias and 1 normal ECG waveform. The keyboard is scanned at the rate of 222 microsecs. The matrix is formed by 3 rows and 8 columns.

Three port pins P1.0, P1.1 and P1.2 are used as return lines for keyboard while ports P1.4, P1.5, P1.6 are read to know which key is pressed. Pins P1.0 to P1.2 are given to 3 to 8 decoder 74HC138 [102].

Keyboard functions

C	B	A	COLUMN ENABLED
P1.2	P1.1	P1.0	
0	0	0	Column No. 0
0	0	1	Column No. 1
0	1	0	Column No. 2
0	1	1	Column No. 3
1	0	0	Column No. 4
1	0	1	Column No. 5
1	1	0	Column No. 6
1	1	1	Column No. 7

When a key closure is detected a zero is read on the corresponding input pin of Microcontroller. Depending on hardware, the keycodes obtained are :

VPB1	60 H	VENTRICULAR FIBRILLATION	63 H
MUTIFOCAL RUN	50 H	ATRIAL FIBRILLATION	53H
TACHYCARDIA	30 H	FUSION BEAT	33 H
MISSED BEAT	61 H	HEART BLOCK	64H
BRADYCARDIA	51 H	PACED	54 H
NORMAL	31H	COUPLET (pair)	34 h
ASYSTOL	62 H	VPB 2	65 H
R ON T WAVE	52 h	VENTRICULAR TACHYCARDIA	55 H
RUN	32 H	BIGEMINY	35 H

4.2.4.2 Display section

There are 18 LEDs used, which will be placed near to the corresponding name of the arrhythmia hence a single LED will glow on the front panel when a particular arrhythmia is selected.

To have 18 LEDs output pins, only 5 port pins i.e. P2.0, P2.1, P2.2, P2.3 and P2.4 are used and which are given to 4 to 16 decoder 74HC154 and 74 HC 138 (3 to 8 decoder) so that 24 pins are available, out of which the first pin is ignored and rest are connected with LEDs in common anode configuration. These port pins are given to input pins of buffer IC 74 HC 245 and after that they are given to decoders. Table 4.1 shows LED configuration.

Table 4.1 LED Configuration

P2.4	P2.3	P2.2	P2.1	P2.0	OUTPUT SELECTED
0	0	0	0	0	IGNORED
0	0	0	0	1	LED 1 = TACHYCARDIA
0	0	0	1	0	LED 2 = NORMAL RHYTHM
0	0	0	1	1	LED 3 = BRADYCARDIA
0	0	1	0	0	LED 4= ASYSTOL
0	0	1	0	1	LED 5= HEART BLOCK
0	0	1	1	0	LED 6= MISSED BEAT
0	0	1	1	1	LED 7= V. FIBRILLATION
0	1	0	0	0	LED 8= V. TACHYCARDIA
0	1	0	0	1	LED 9= BIGEMINY
0	1	0	1	0	LED 10= VPB1
0	1	0	1	1	LED 11= MUTIFOCAL RUN
0	1	1	0	0	LED 12= FUSION BEAT
0	1	1	0	1	LED 13= VPB 2
0	1	1	1	0	LED 14 = COUPLET (pair)
0	1	1	1	1	LED 15 = RUN
1	0	0	0	0	LED 16 = R ON T WAVE
1	0	0	0	1	LED 17 = ATRIAL FIBRILLATION
1	0	0	1	0	LED 18 = PACED RHYTHM

4.3 SOFTWARE

Programming of 8752 microcontroller is done in assembly language. The following session give the details of the steps involved in programming.

4.3.1 Algorithm and flowcharts

8752 microcontroller has two software interrupts T_0, T_1. It is difficult to program using "polling" method keyboard scanning along with outputting display and waveforms. The best method therefore is to use the software timer interrupt provided by the microontroller. Each timer interrupt can be used in mode 0, mode 1, mode 2, or mode 3 operation. The details of the modes of operation is provided in the appendix. Mode 1 is the mode used for our purpose in which the timer register can be loaded as 16 bit data.

Complete data for each waveform is to be outputted in 2msecs. Since all the 9 waveforms are outputted at a time, the timing for each waveform data byte is 2msecs /9 i.e. 222 microsecs. Hence the timer is loaded with a count for 222 microsecs. Timer mode is selected using TMOD register, timer interrupt is selected using TCON register and TH0, TL0 are loaded with the appropriate count. IE register is used to enable the interrupt, since it is started because of TCON register.

4.3.1.4 Algorithm

1. Initialize all ports of 8752 microcontroller.
2. Initialize timer interrupt TO.
3. Start timer and enable timer interrupt.
4. Wait till interrupt comes.
5. In timer interrupt routine, scan the keyboard.
6. If new keypress is detected find the keycode.
7. If no keypress is there continue the previous operation.
8. Depending on the keycode, goto respective routine.

9. In arrhythmia routine, check for initialization flag and if not set do the initialization of the memory pointer, counter for data bytes and scan lines

10. Fetch appropriate byte from the respective memory location and output to DAC port.

11. Output scan lines to enable the analog Demux switches.

12. Increment memory pointer and scan line counter for next data.

13. Check for scan lines, if greater than 9 reinitialize it to zero.

14. Counter is decremented by 1, and if zero clear the initialization flag.

15. Return from interrupt.

Depending on the arrhythmia the operations are carried out accordingly, for eg. in Heart block there is a normal ECG waveform followed by a delay and occurrence of only P wave (QRS and T waves are absent) again there is a delay and a normal pattern. This process is repeated. Depending on such operations certain flags are set for carrying out intermediate operations and are checked to continue the process.

Program is written in assembly language of 8752 microcontroller using Norton editor, assembled using Cross 16 Assembler and using HEXOBJ software for obtaining the object code. This data is then loaded into the 8752 EPROM using the EPROM programmer.

4.3.1.2 Flowcharts

Refer figures 4.5, 4.6, 4.7 for program flowcharts .

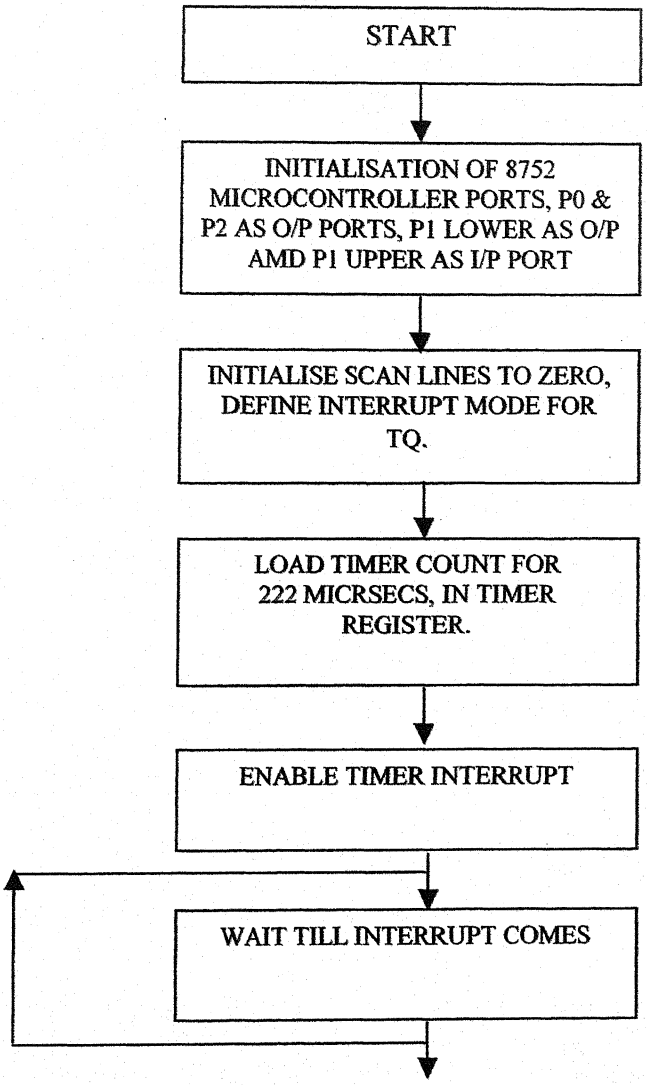

Fig. 4.5 : Flowchart – Initialisation Program of ECG/Arrhythmia Simulator using Microcontroller

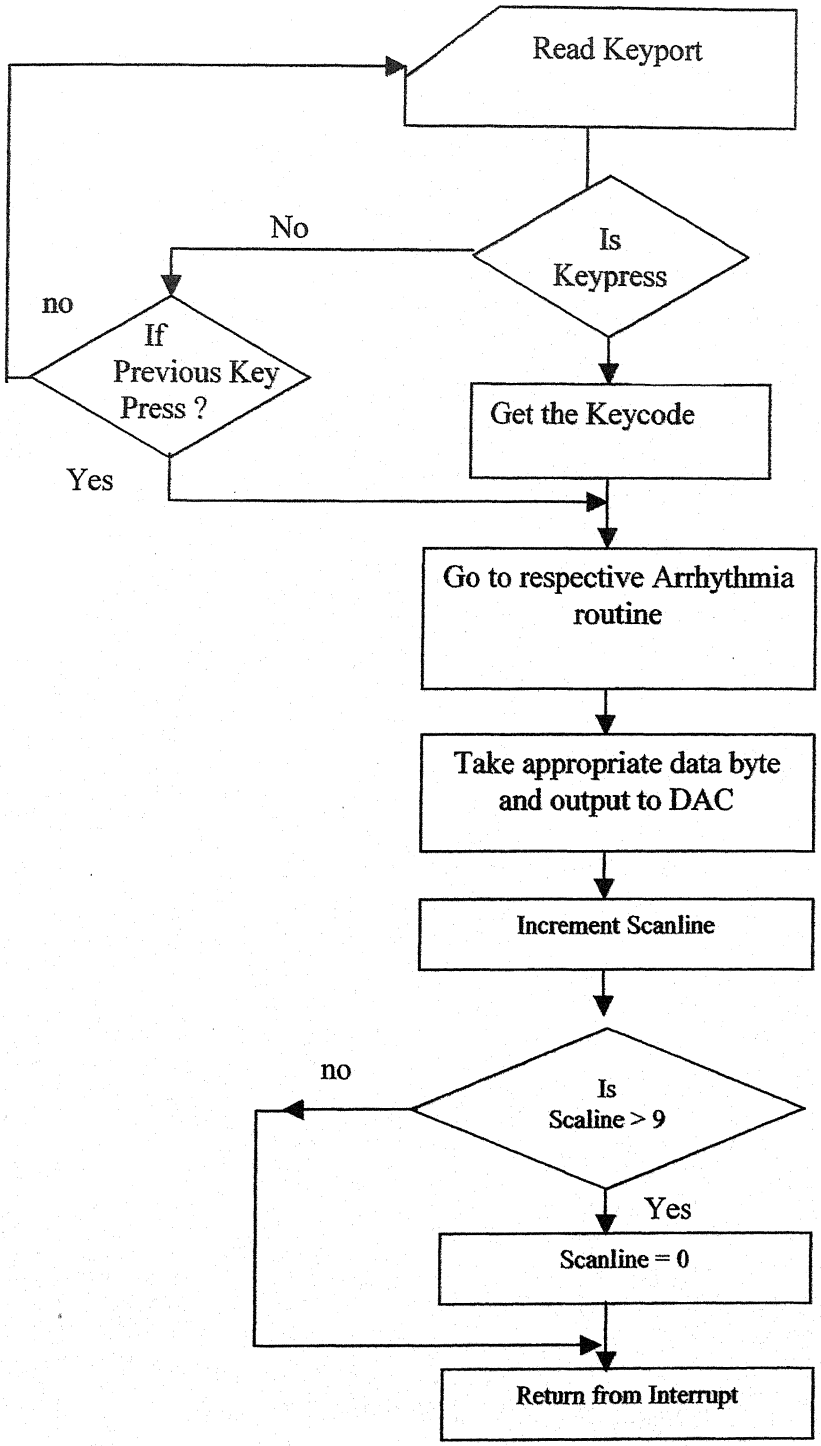

Fig. 4.6 : Generalised Flowchart To detect Arrhythmia using microcontroller

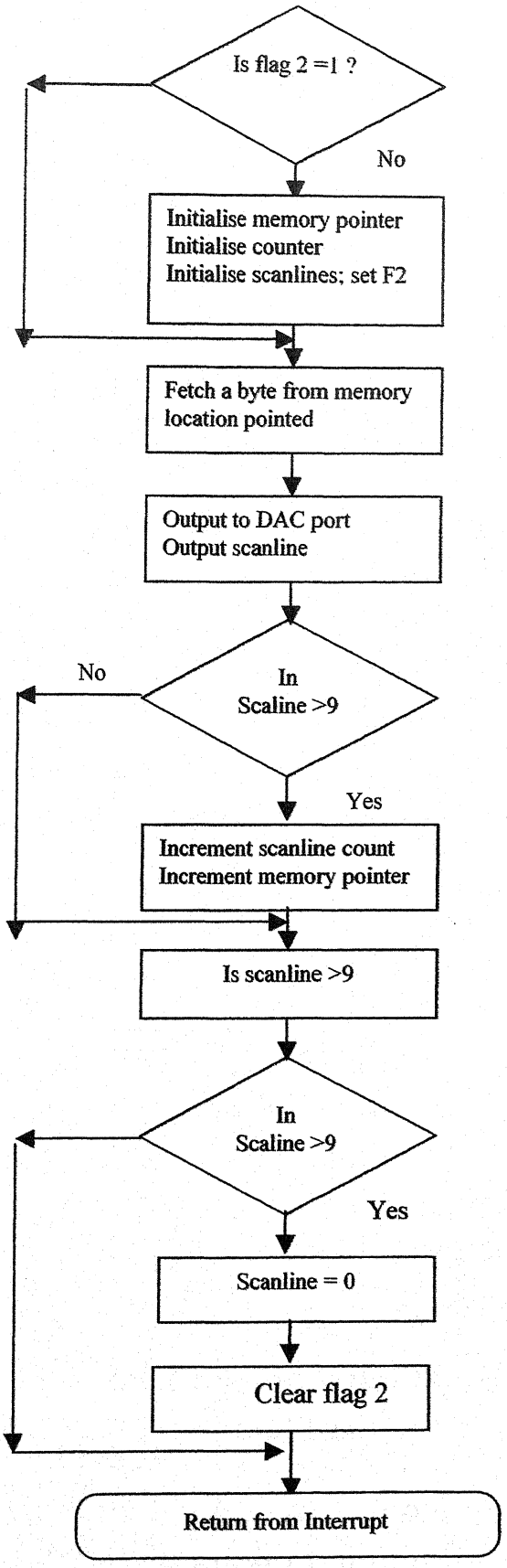

Fig. 4.7 : Flowchart of arrhythmia (Tachycardia) using microcontroller

4.3.2 HEX CODES USED IN SOFTWARE

Keycode function	:	Keycode function	: keycode function
	:		:
30h- tachycardia	:	50h- multfocal run	: 60h- VPB 1
31h- normal	:	51h- bradycardia	: 61h- Missed beat
32h- run	:	52h- R on T wave	: 62h- Asystole
33h fusionBeat	:	53h- Atrial Fibrillation	63h-Ventricular fibrillation
43h -couplet	:	54h -Paced Rhythm	: 64h- Heart Block
35h- bigeminy	:	55h- Ventricular tachycardia	: 65h- VPB 2

port identification

P0- digital data to DAC

P1- lower port for scan lines

P1- upper port for keyboard read

P2- display port

flags used here :

bit 00h & 01h as f0and f1 respectively for keyboard

bit 02h as initialisation flag for all waveforms

bits 03h, 04h, 05h, 06h as intermediate flags for internal operation

4.3.3 LOOK UP TABLES

The data stored in the EPROM of 8752 for different arrhythmias are given in following tables.

Table 4.2 : The for normal ECG signal

Org 0800h

```
dfb     75h,7dh,64h,7bh,80h,66h,60h,66h,66h
dfb     75h,7dh,63h,7bh,80h,66h,60h,66h,66h
dfb     75h,7dh,63h,7bh,80h,66h,5fh,65h,66h
```

```
dfb     75h,7dh,62h,7bh,80h,66h,5fh,65h,66h
dfb     74h,7ch,62h,7bh,1fh,65h,5eh,64h,65h
dfb     74h,7ch,61h,7bh,7fh,65h,5eh,63h,65h
dfb     74h,7ch,61h,7ah,7fh,64h,5dh,62h,64h
dfb     74h,7ch,60h,7ah,7fh,64h,5dh,62h,64h
dfb     73h,7bh,60h,7ah,7fh,63h,5ch,61h,63h
dfb     73h,7bh,5fh,7ah,7fh,63h,5ch,61h,63h
dfb     73h,7bh,5eh,7ah,7eh,62h,5ch,60h,62h
dfb     73h,7bh,5eh,7ah,7eh,62h,5ch,5fh,62h
dfb     73h,7bh,5dh,7ah,7eh,61h,5bh,5eh,62h
dfb     73h,7bh,5dh,7ah,7eh,61h,5bh,5eh,62h
dfb     72h,7ah,5ch,7ah,7eh,60h,5ah,5dh,61h
dfb     72h,7ah,5ch,79h,7eh,60h,_ah,5dh,61h
dfb     72h,7ah,5bh,79h,7eh,5fh,5ah,5ch,61h
dfb     72h,7ah,5bh,79h,7eh,5eh,5ah,5ch,61h
dfb     72h,7ah,5ah,79h,7eh,5dh,59h,5bh,60h
dfb     71h,7ah,5ah,19h,7eh,5ch,59h,5bh,60h
dfb     71h,79h,59h,79h,7eh,5bh,58h,5ah,5fh
dfb     71h,19h,59h,78h,7eh,5ah,58h,5ah,5fh
dfb     71h,79h,58h,78h,7dh,59h,58h,59h,5dh
dfb     70h,79h,58h,78h,7dh,59h,58h,59h,5dh
dfb     70h,79h,57h,78h,7dh,59h,58h,59h,5ch
dfb     70h,19h,57h,78h,7dh,59h,58h,59h,5ch
dfb     70h,79h,56h,78h,7dh,58h,58h,5ah,5bh
dfb     70h,79h,56h,78h,7dh,56h,58h,5ah,5bh
dfb     70h,79h,55h,78h,7dh,55h,58h,5ah,59h
dfb     70h,79h,55h,78h,7dh,54h,58h,5ah,59h
dfb     70h,78h,54h,78h,7dh,54h,58h,5bh,5ah
dfb     70h,78h,54h,78h,7dh,53h,58h,5bh,5ah
dfb     6fh,78h,54h,78h,7dh,51h,59h,5ch,5bh
dfb     6fh,78h,54h,77h,7dh,50h,59h,5ch,5bh
dfb     6fh,78h,54h,77h,7ch,50h,5ah,5dh,5ch
```

```
dfb     6fh,18h,54h,77h,7ch,4fh,5ah,5dh,5ch
dfb     6eh,78h,53h,77h,7ch,4fh,5ah,5eh,5dh
dfb     6eh,78h,53h,77h,7ch,4eh,5ah,5eh,5dh
dfb     6dh,78h,53h,77h,7ch,4dh,5ah,5fh,60h
dfb     6dh,78h,53h,71h,1ch,4ch,5ah,5fh,60h
dfb     6dh,78h,53h,77h,7ch,4ch,5bh,60h,61h
dfb     6dh,78h,53h,77h,7ch,4dh,5bh,60h,61h
dfb     6dh,78h,53h,77h,7ch,4dh,5ch,61h,62h
dfb     6dh,78h,53h,17h,7ch,4eh,5ch,61h,62h
dfb     6dh,79h,53h,77h,7ch,4eh,5dh,62h,63h
dfb     6dh,79h,53h,77h,7ch,4fh,5dh,63h,63h
dfb     6dh,79h,54h,77h,7ch,4fh,5eh,64h,64h
dfb     6dh,79h,54h,77h,7ch,50h,5eh,64h,64h
dfb     6fh,7ah,59h,78h,7dh,54h,63h,69h,67h
dfb     6fh,7ah,59h,78h,7dh,55h,63h,69h,67h
dfb     6fh,7ah,5ah,78h,7dh,55h,64h,6ah,68h
dfb     6fh,7ah,5bh,78h,7dh,56h,64h,6ah,68h
dfb     70h,7ah,5ch,78h,7eh,57h,65h,6bh,68h
dfb     70h,7ah,5ch,78h,7eh,58h,65h,6ch,68h
dfb     70h,7bh,5dh,78h,7eh,59h,66h,6dh,69h
dfb     70h,7bh,5eh,79h,7eh,5ah,66h,6dh,69h
dfb     71h,7bh,5fh,79h,7eh,5ah,67h,6eh,6ah
dfb     71h,7bh,5fh,79h,7eh,5bh,67h,6eh,6ah
dfb     71h,7ch,60h,79h,7eh,5ch,68h,6fh,6ah
dfb     71h,7ch,61h,79h,7eh,5dh,68h,6fh,6ah
dfb     72h,7ch,62h,79h,7eh,5dh,69h,70h,6bh
dfb     72h,7ch,62h,79h,7eh,5eh,69h,70h,6bh
dfb     73h,7ch,63h,79h,7eh,5fh,69h,71hi6dh
dfb     73h,7ch,63h,7ah,7eh,60h,69h,72h,6dh
dfb     74h,7dh,64h,7ah,7eh,60h,6ah,73h,6eh
dfb     74h,7dh,65h,7ah,7eh,61h,6ah,74h,6eh
dfb     74h,7dh,66h,7ah,7eh,61h,6bh,75h,6fh
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dfb     74h,7dh,67h,7ah,7eh,62h,6ch,77h,6fh
dfb     75h,7dh,67h,7ah,7fh,63h,6dh,79h,70h
dfb     75h,7dh,68h,7bh,7fh,64h,6dh,7bh,70h
dfb     76h,7eh,68h,7bh,7fh,65h,6eh,7ch,71h
dfb     76h,7eh,69h,7bh,7fh,66h,6fh,78h,71h
dfb     77h,7eh,6ah,7bh,7fh,66h,70h,75h,72h
dfb     77h,7eh,6bh,7bh,7fh,66h,71h,73h,72h
dfb     78h,7fh,6bh,7bh,7fh,67h,73h,6fh,75h
dfb     78h,7fh,6ch,7ch,7fh,68h,6fh,64h,75h
dfb     78h,7fh,6ch,7ch,7fh,69h,6ch,59h,7dh
dfb     78h,7fh,6dh,7ch,7fh,6bh,66h,4dh,7dh
dfb     79h,7fh,6eh,7ch,7fh,6ch,60h,42h,7ch
dfb     79h,7fh,6fh,7dh,7fh,6dh,5ch,38h,7ch
dfb     7ah,80h,70h,7dh,7fh,6eh,58h,2fh,77h
dfb     7ah,80h,71h,7eh,7fh,70h,43h,25h,73h
dfb     7bh,7fh,72h,7eh,80h,72h,4ch,lbh,60h
dfb     7ch,7fh,73h,7eh,80h,73h,46h,17h,60h
dfb     7dh,7eh,71h,7eh,80h,6bh,40h,13h,40h
dfb     7dh,7eh,6dh,7fh,80h,60h,35h,11h,40h
dfb     7eh,7ch,68h,7fh,80h,53h,2ah,0h,2ch
        7eh,7ch,63h,7fh,80h,49h,18h,0h,2ch
dfb     7fh,78h,60h,7fh,81h,40h,0h,0h,lfh
dfb     7eh,78h,43h,80h,82h,26h,0h,0h,lfh
dfb     7bh,70h,30h,80h,81h,19h,0h,0h,17h
dfb     79h,70h,22h,7eh,80h,13h,0h,0h,17h
dfb     76h,6ch,lbh,7dh,7fh,0h,0h,l0,13h
dfb     73h,6ch,17h,7bh,7eh,0h,0h,18h,13h
dfb     6bh,6ah,13h,76h,7dh,0h,0h,20h,19h
dfb     68h,6ah,0h,71h,7bh,03h,0034h,19h
dfb     63h,69h,0h,70h,79h,0h,0h,49h,2ah
dfb     6dh,79h,5bh,73h,7dh,53h,4ah,53h,52h
dfb     6dh,79h,5ch,73h,7dh,54h,4bh,54h,54h
```

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dfb     6dh,79h,5dh,73h,7dh,55h,4ch,54h,54h
dfb     6eh,7ah,5dh,73h,7dh,56h,4dh,55h,56h
dfb     6eh,7ah,5eh,73h,7dh,57h,4dh,57h,56h
dfb     6fh,7ah,5fh,73h,7dh,58h,4eh,58h,59h
dfb     6fh,7ah,61h,73h,7dh,59h,4fh,58h,59h
dfb     6fh,7bh,61h,73h,7dh,5ah,50h,59h,5bh
dfb     6fh,7bh,62h,73h,7dh,5bh,52h,5ah,5bh
dfb     70h,7bh,63h,73h,7eh,5ch,53h,5bh,5ch
dfb     70h,7bh,63h,73h,7eh,5dh,54h,5bh,5ch
dfb     70h,7ch,64h,74h,7eh,5eh,55h,5ch,5dh
dfb     70h,7ch,64h,74h,7eh,60h,57h,5dh,5dh
dfb     71h,7ch,65h,74h,7eh,60h,59h,5eh.5eh
dfb     71h,7ch,65h,74h,7eh,61h,5bh,5fh,5eh
dfb     72h.7ch,66h,75h.7eh,61h,5dh.60h,60h
dfb     72h,7ch,66h,75h,7eh,62h,5fh,60h,60h
dfb     72h,7ch,67h,75h,7eh,62h,60h,61h,61h
dfb     72h,7ch,67h,75h,7eh,63h,60h,61h,61h
dfb     73h,7dh,66h,75h,7fh,63h,61h.62h,62h
dfb     73h,7dh,68h,75h,7fh,64h,61h,62h,62h
dfb     73h,7dh,68h,76h,7fh,64h,62h,63h,63h
dfb     73h,7dh,68h,76h,7fh,65h,62h,64h,63h
dfb     73h,7dh,6Bh,77h,7fh,65h,63h,65h,64h
dfb     73h,7dh,68h,77h,7fh,66h,63h,65h,65h
dfb     73h,7dh,67h,78h,7fh,66h,63h,66h,66h
dfb     73h,7dh,67h,78h,7fh,66h,63h,66h,66h
dfb     74h,7dh,67h,78h,7fh,66h,64h,67h,67h
dfb     74h,1dh,67h,78h,7fh,67h,64h,67h,67h
dfb     74h,7eh,66h,79h,7fh,67h,64h,67h,67h
dfb     74h,7eh,66h,79h,7fh,67h,64h,67h,67h
dfb     74h,7eh,66h,7ah,80h,68h,63h,68h,67h
dfb     74h,7eh,66h,7ah,80h,68h,63h,68h,67h
```

```
dfb     74h,7eh,65h,7ah,80h,68h,63h,68h,67h
dfb     74h,7eh,65h,7ah,80h,68h,63h,68h,67h
dfb     74h,7dh,64h,7bh,80h,67h,62h,67h,66h
dfb     74h,7dh,64h,7bh,80h,67h,61h,67h,66h
```

Table 4.3 : Data for vpb1

```
        org     10cbh
dfb             00h, 00h, 00h
dfb             7eh, 80h, 6eh
dfb             7ah, 80h, 6eh
dfb             76h, 7fh, 65h
dfb             73h, 7eh, 5dh
dfb             70h, 7eh, 57h
dfb             6dh, 7dh, 50h
dfb             6ah, 7bh, 4bh
dfb             68h, 79h, 46h
dfb             65h, 77h, 41h
dfb             62h, 76h, 3ch
dfb             5fh, 74h, 35h
dfb             5dh, 73h, 30h
dfb             91h, 88h, 0a5h
dfb             92h, 88h, 0a6h
dfb             92h, 88h, 0a7h
dfb             91h, 87h, 0a6h
dfb             91h, 87h, 0a5h
dfb             91h, 87h, 0a4h
dfb             90h, 87h, 0a4h
dfb             8fh, 87h, 0a3h
dfb             8eh, 86h, 0a2h
dfb             8eh, 86h, 0a1h
dfb             8dh, 86h, 0a0h
```

```
dfb     8dh, 85h, 9fh
dfb     8ch, 85h, 9eh
dfb     8bh, 85h, 9dh
dfb     8ah, 85h, 9bh
dfb     89h, 84h, 9ah
dfb     88h, 84h, 98h
dfb     88h, 84h, 95h
dfb     87h, 84h, 93h
dfb     87h, 83h, 91h
dfb     86h, 83h, 90h
dfb     86h, 83h, 8dh
dfb     85h, 83h, 8ch
dfb     85h, 82h, 8bh
dfb     84h, 82h, 88h
dfb     84h, 82h, 87h
dfb     83h, 82h, 86h
dfb     83h, 82h, 84h
dfb     82h, 82h, 83h
dfb     82h, 82h, 82h
dfb     82h, 81h, 81h
dfb     82h, 81h, 80h
dfb     81h, 81h, 7eh
dfb     81h, 81h, 7dh
dfb     80h, 81h, 7dh
dfb     80h, 81h, 7ch
dfb     80h, 81h, 7bh
dfb     80h, 81h, 7ah
dfb     7fh, 80h, 79h
dfb     7fh, 80h, 78h
dfb     7fh, 80h, 78h
dfb     7fh, 80h, 77h
```

```
dfb     7fh, 80h, 76h
dfb     7eh, 80h, 75h
dfb     7eh, 80h, 74h
dfb     7eh, 80h, 74h
dfb     7eh, 80h, 73h
```

Table 4.4 : Data for atrial firbrillation

```
        org     1228h
dfb     7dh, 80h, 73h
dfb     76h,7ch,60h
```

Table 4.5 : Data for ventricular fibrillation

```
        org     1240h
dfb     7eh, 80h, 77h
dfb     7dh, 80h, 67h
dfb     7ch, 7fh, 6bh
dfb     7ah, 7eh, 69h
dfb     79h, 7dh, 65h
dfb     77h, 7dh, 60h
dfb     75h, 7ch, 59h
dfb     73h, 7ch, 54h
dfb     71h, 7bh, 50h
dfb     70h, 7ah, 4eh
dfb     6fh, 79h, 4ch
dfb     6dh, 79h, 4bh
dfb     6bh, 79h, 4ah
dfb     69h, 78h, 49h
dfb     68h, 78h, 48h
```

```
dfb     68h, 77h, 49h
dfb     67h, 77h, 49h
dfb     68h, 77h, 4ah
dfb     69h, 77h, 4bh
dfb     6ah, 77h, 4eh
dfb     6bh, 76h, 50h
dfb     6bh, 76h, 50h
dfb     6bh, 77h, 52h
dfb     6bh, 77h, 53h
dfb     6bh, 77h, 54h
dfb     6ch, 78h, 56h
dfb     6dh, 78h, 58h
dfb     6eh, 78h, 59h
dfb     6fh, 78h, 5bh
dfb     70h, 79h, 5dh
dfb     70h, 79h, 60h
dfb     71h, 7ah, 63h
dfb     72h, 7ah, 66h
dfb     73h, 7ah, 69h
dfb     73h, 7bh, 6bh
dfb     74h, 7bh, 6ch
dfb     75h, 7ch, 6eh
dfb     76h, 7ch, 70h
dfb     77h, 7dh, 75h
dfb     78h, 7dh, 78h
dfb     78h, 7eh, 79h
dfb     79h, 7eh, 7bh
dfb     78h, 7fh, 7dh
dfb     76h, 80h, 7eh
dfb     74h, 81h, 7fh
dfb     72h, 82h, 80h
```

```
dfb     6eh, 82h, 80h
dfb     6dh, 82h, 7fh
dfb     6ch, 83h, 7dh
dfb     6ch, 82h, 7ch
dfb     6bh, 82h, 7ah
dfb     7ch, 7dh, 67h
dfb     7dh, 7dh, 65h
dfb     7dh, 7ch, 63h
dfb     7eh, 7bh, 62h
dfb     7eh, 7bh, 61h
dfb     7fh, 7bh, 60h
dfb     80h, 7ah, 60h
dfb     7fh, 7ah, 61h
dfb     7fh, 7bh, 62h
dfb     7eh, 7bh, 65h
dfb     7dh, 7ch, 66h
dfb     7ch, 7ch, 69h
dfb     7bh, 7dh, 6bh
dfb     7ah, 7dh, 6dh
dfb     7ah, 7dh, 6fh
dfb     79h, 7eh, 71h
dfb     79h, 7eh, 74h
dfb     78h, 7fh, 79h
dfb     7ah, 80h, 7bh
dfb     7bh, 80h, 7dh
dfb     7bh, 81h, 7eh
dfb     7ch, 82h, 7eh
dfb     7ch, 84h, 7fh
dfb     7dh, 85h, 80h
dfb     7dh, 86h, 7fh
dfb     7eh, 85h, 7eh
```

```
dfb     7eh, 85h, 7dh
dfb     7fh, 84h, 7ch
dfb     7fh, 84h, 7bh
dfb     80h, 83h, 79h
dfb     7fh, 83h, 78h
dfb     7eh, 82h, 77h
dfb     7eh, 82h, 75h
dfb     7fh, 81h, 74h
dfb     7fh, 80h, 73h
dfb     7eh, 80h, 73h
```

4.4 PCB LAYOUT AND FABRICATION

The circuit is split into two sections :

1. The microcontroller and analog section
2. Keyboard and display section

PCBs are designed using "Smartwork" package which provides the facility to make artworks of double sided PCBs. Proper sizes for the boards are selected, the solder side , the component side, and the silk side are plotted and given for PCB fabrication.

4.4.1 MICROCONTROLLER AND ANALOG SECTION

This PCB includes the microcontroller and all the analog output circuitry and the power supply and three connectors for 1mV, 1V, and third for interfacing the keyboard display board. Refer Figures 4.8,4.9,4.10 for PCB layout of analog section of ECG/Arrhythmia simulator using microcontroller. Figure 4.8 shows solder side, figure 4.9 shows component side and figure 4.10 shows silks screen of PCG layout of analog section of ECG/Arrhythmia simulator using microcontroller.

4.4.2 KEYBOARD AND DISPLAY SECTION

This consists the keyboard matrix of 20 keys, 18 LEDs display and a connector to interface with the main board. Refer Figures 4.11, 4.12,4.13 for PCB Layout of keyboard and display section of ECG/ Arrhythmia simulator using microcontroller. Figure 4.11 shows solder side, figure 4.12 shows component side and figure 4.13 shows silks screen of PCB Layout of keyboard and display section of ECG/ Arrhythmia simulator using microcontroller .

4.5 TESTING OF PCBs

Once the PCBs were obtained, they were checked for the continuity, shortings etc. The assembly of the circuit is started part by part. First the DAC circuit is assembled on breadboard to determine the values of circuit components. When it was confirmed with satisfactory results. It was assembled on the PCB. Similarly the DEMUX Ics and its analog circuit was implemented and assembled.

Step by step all the circuits were assembled and checked. This confirmed the proper functioning of the hardware section on the main board.

4.5.1 TESTING OF POWER SUPPLY

First the power supply circuit is implemented and checked. The output of the power supply is +5V, -5V, ripple rejection is 70 dB.

4.5.2 TESTING OF MICROCONTROLLER CIRCUIT

The microcontroller circuit is assembled on breadboard, the crystal oscillator circuit is connected and the test program is written and run to output a square wave on each port pin at fixed timer operation of 222 microseconds duration. This confirmed proper operation of the microcontroller in generating ALE signal. The 40 pin microcontroller socket and the oscillator circuit is implemented on the PCB.

1X checkplot 1 Jan 2006 05:49:02

d : sa1 . pcb

v1.2 r2 holes : 678 Solder side

approximate size : 5.50 by 6.45 inches

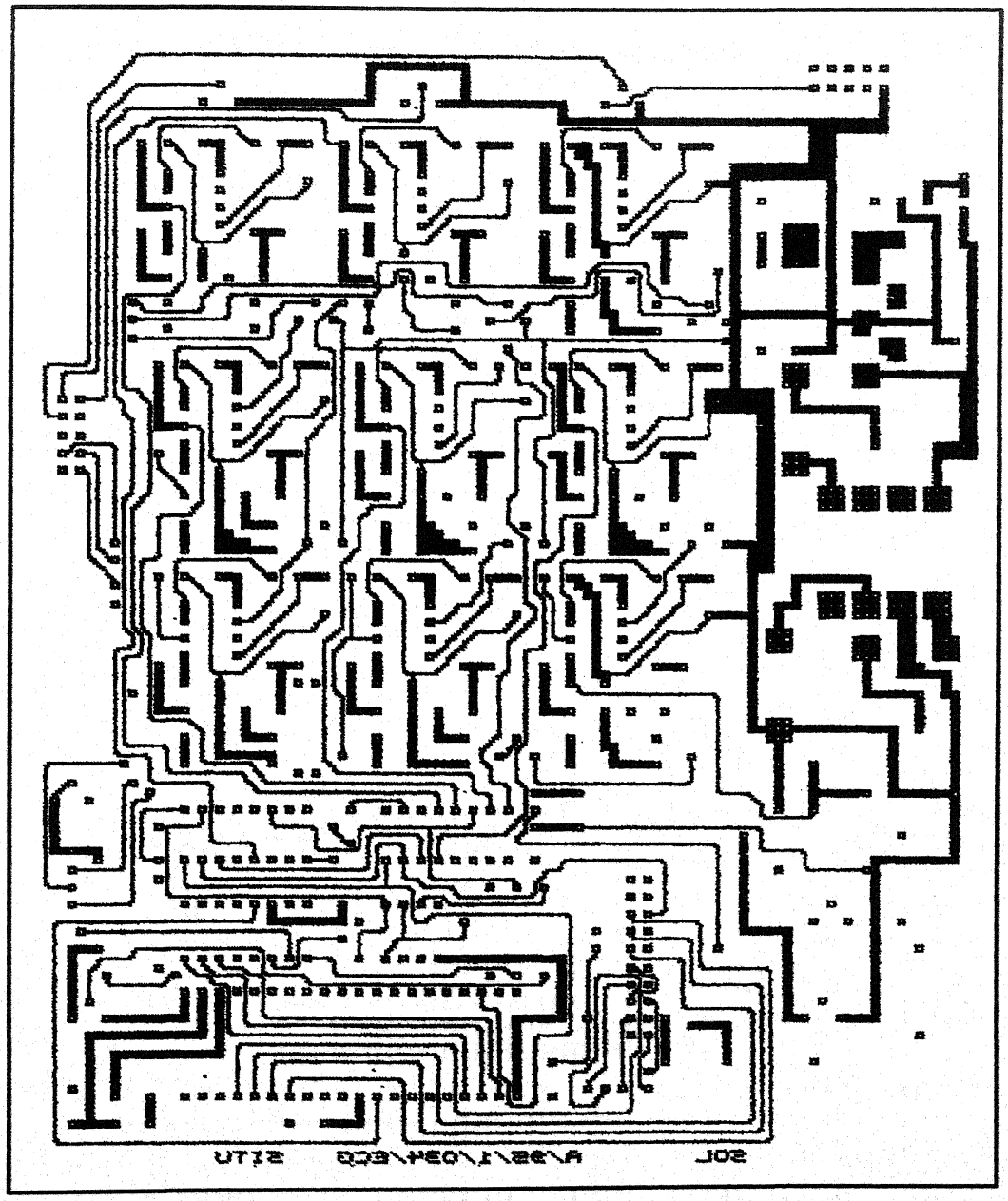


Fig. 4.8 : PCB layout solder side of ECG/Arrhythmia simulator using microcontroller

1X checkplot 1 Jan 2006 06 : 01 : 15

d : sa1 . pcb

v1.2 r2 holes : 678 Component side

approximate size : 5.50 by 6.45 inches

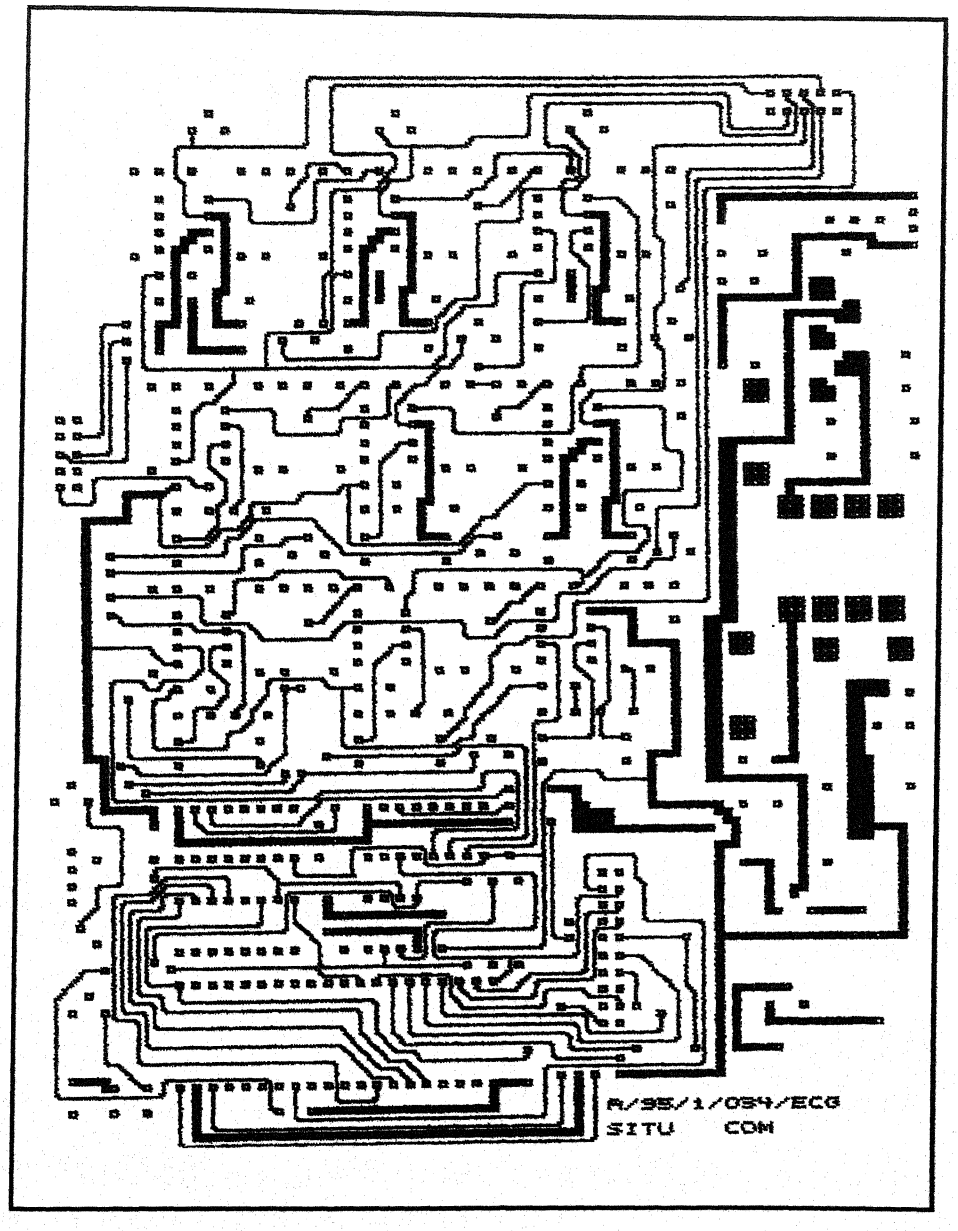


Fig. 4.9 : PCB layout component side of ECG/Arrhythmia simulator using microcontroller

1X checkplot 1 Jan 2006 06 : 05: 08

d : sa1 . pcb

v1.2 r2 holes : 678 Silk Screen side

approximate size : 5.50 by 6.45 inches

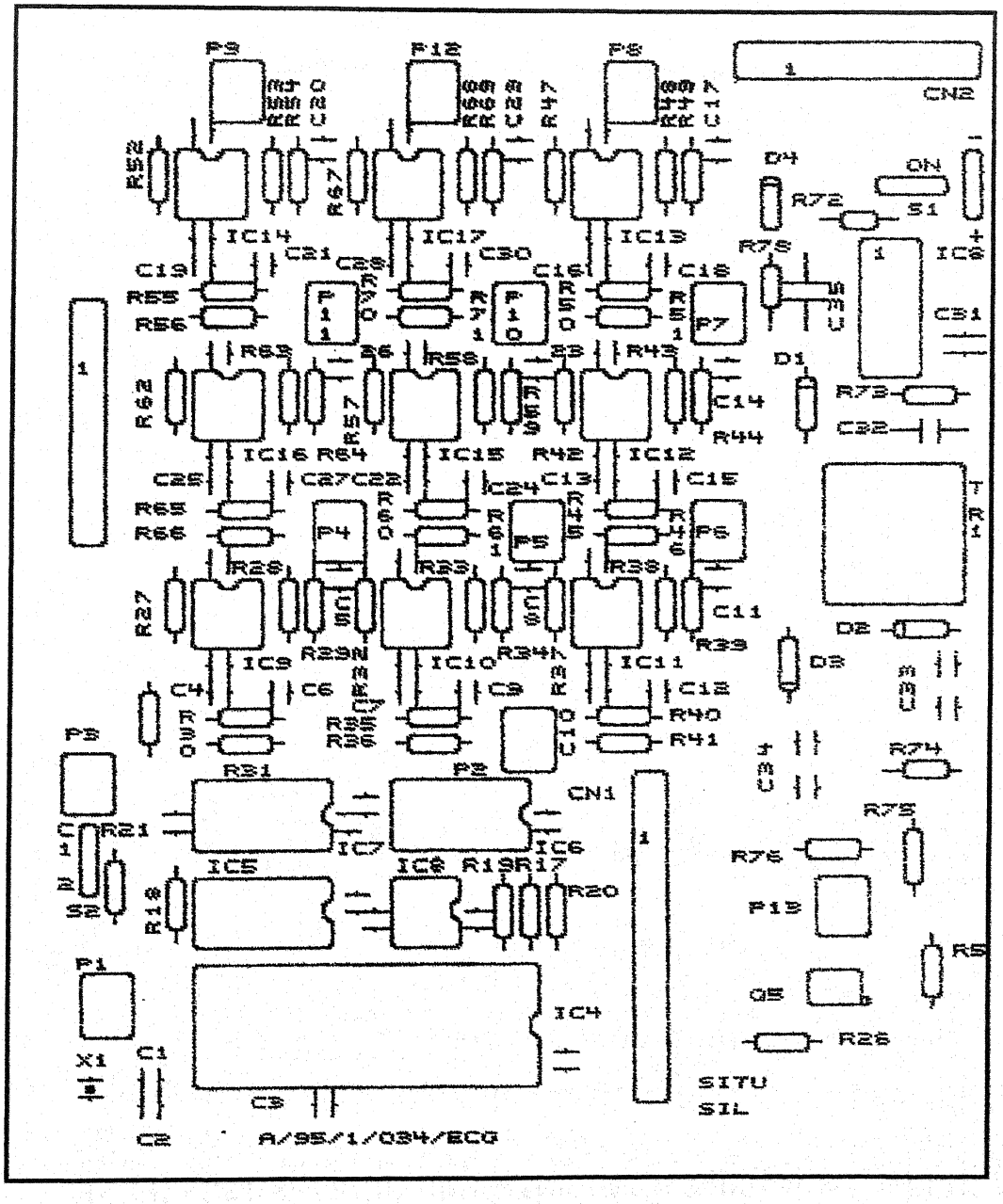


Fig. 4.10 PCB layout Silk Screen side of ECG/Arrhythmia simulator using microcontroller

1X checkplot 1 Jan 2006 06 : 08: 19

d : sa2 . pcb

v1.2 r2 holes : 218 Silk Screen side

approximate size : 3.75 by 4.60 inches

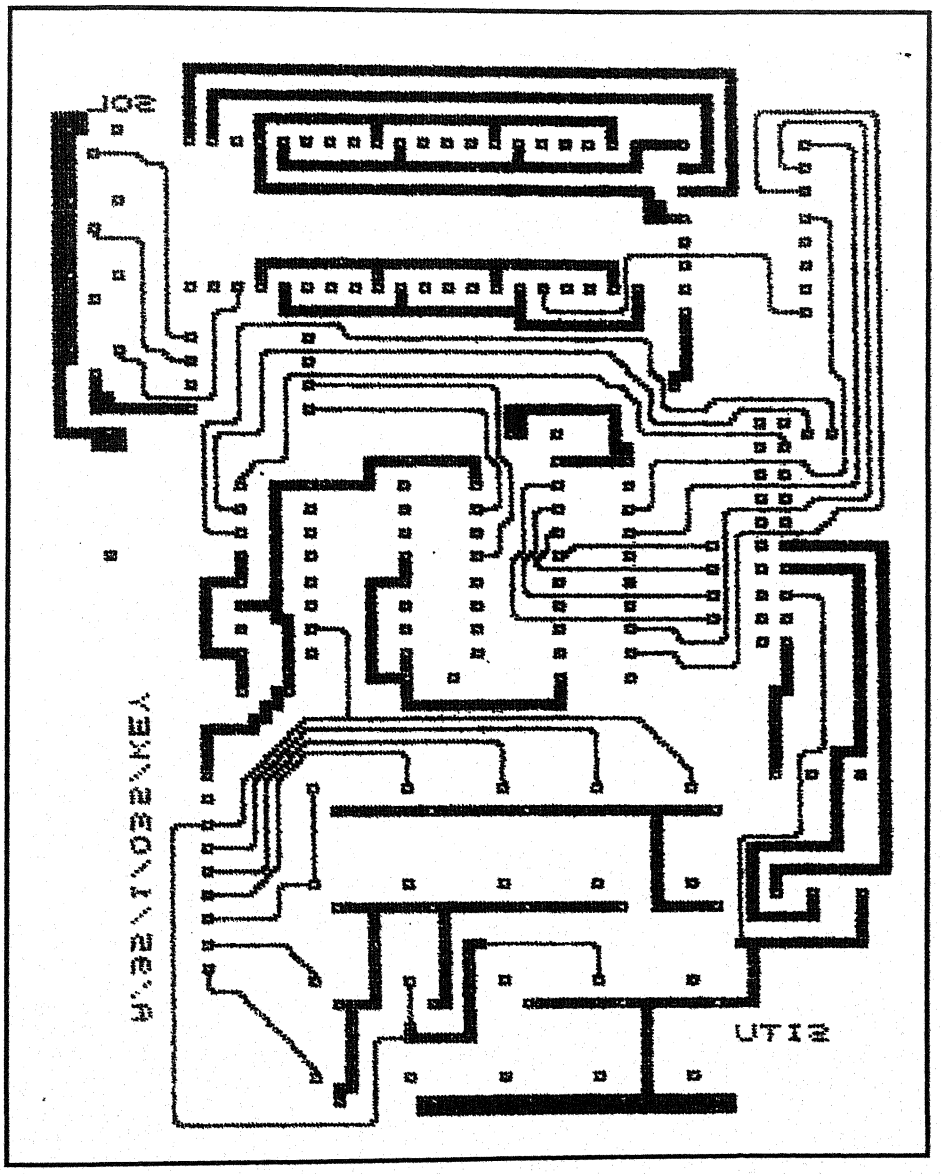


Fig. 4.11 PCB layout Solder side of keyboard and display section of ECG/ Arrhythmia simulator using microcontroller.

1X checkplot 1 Jan 2006 06 : 10: 29

d : sa2 . pcb

v1.2 r2 holes : 218 Component side

approximate size : 3.75 by 4.60 inches

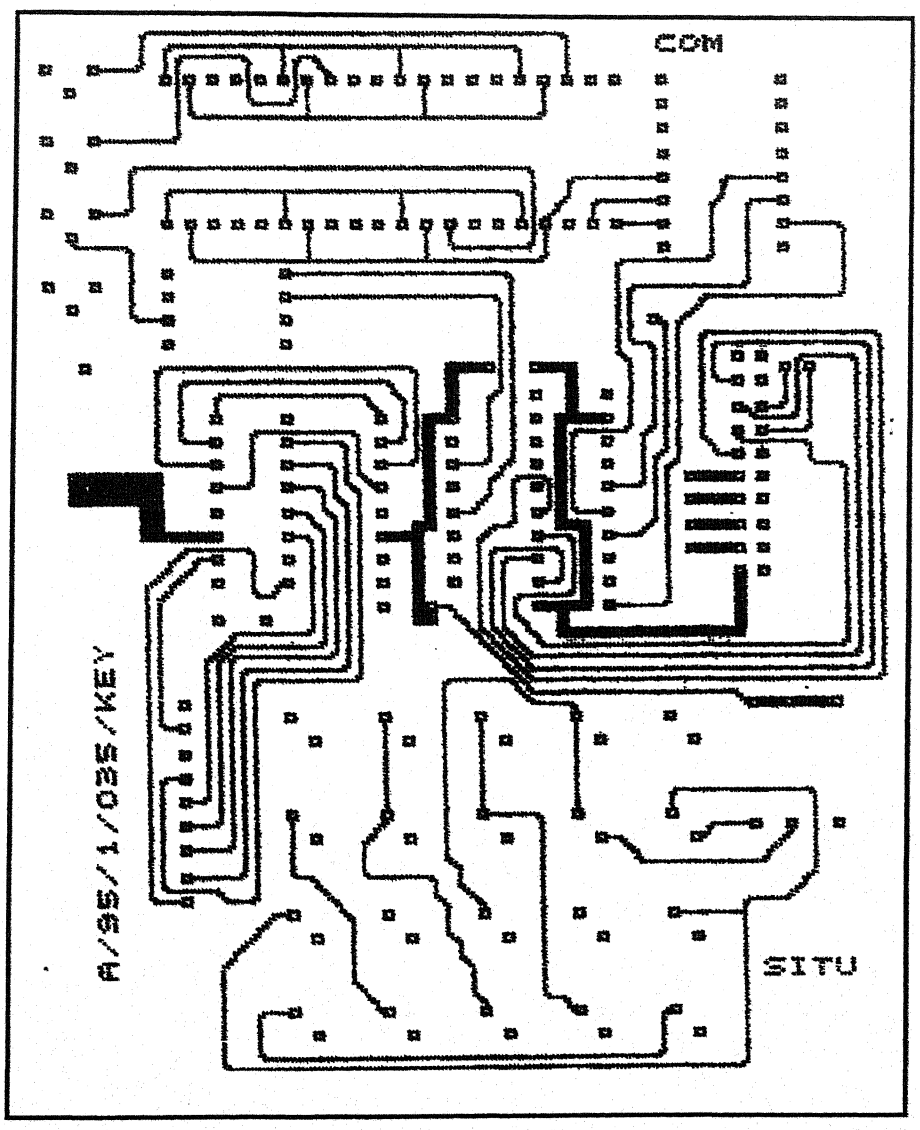


Fig. 4.12 PCB layout - Component side of keyboard and display section of ECG/ Arrhythmia simulator using microcontroller.

1X checkplot 1 Jan 2006 06 : 12 : 50

d : sa2 . pcb

v1.2 r2 holes : 218 Silk Screen

approximate size : 3.75 by 4.60 inches

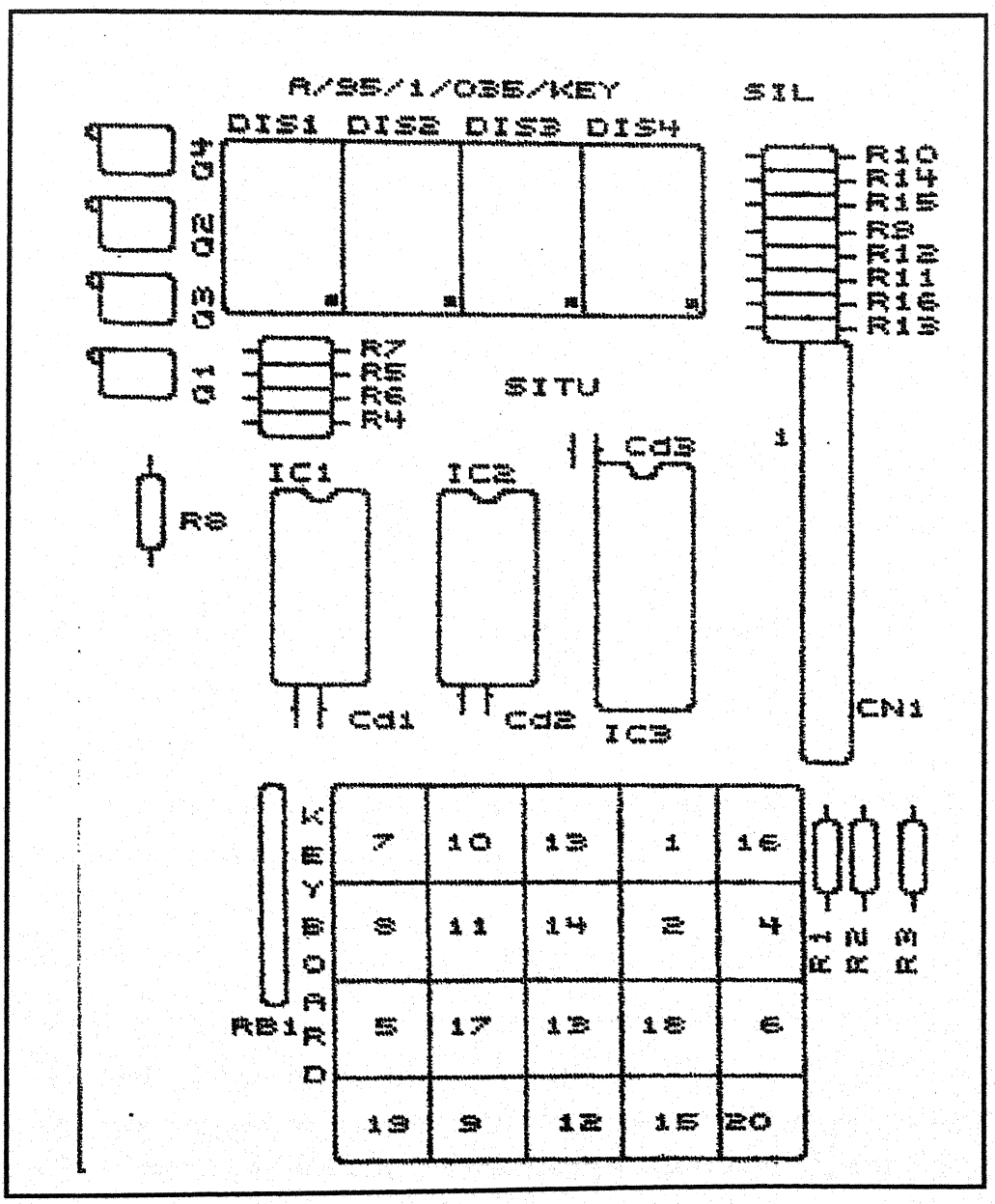


Fig. 4.13 PCB layout – Silk Screen of keyboard and display section of ECG/ Arrhythmia simulator using microcontroller.

4.5.3 TESTING OF KEYBOARD AND DISPLAY

The display LEDs and the switches are checked whether they are working properly or not. On confirming their operation they were assembled on the PCB.

4.5.4 SOFTWARE IMPLEMENTATIONS

The steps followed for software implementations are :

1. Program is written for every arrhythmia.
2. One by one all the arrhythmias are checked.
3. Necessary changes are made in the data for proper outputs.
4. All the nine waveforms (signals) are observed on the storage oscilloscope.
5. Differential amplifier was implemented on the breadboard for observing lead I, lead II, lead III, aV_R, aV_L, aV_F signals.
6. Once all the arrhythmias are checked and data was confirmed, it was stored in the final look up table in the EPROM.
7. Keyboard detection routine was written and all the 18 arrhythmia routines are assembled in file. Depending on the key –press, the routine is called and the operation is carried out on interrupt generation at 222microseconds.
8. Simultaneously the appropriate LED is glown on the display board.

Once the complete software was over, it was loaded in the 8752 EPROM, and working of the hardware is checked on storage oscilloscope.

The instrument was checked at Bhabha hospital, Bandra, Mumbai on ECG machine for its proper functioning.

4.6 RESULTS & CONCLUSION

Once the operation of the hardware is confirmed, the unit is fabricated and assembled. The assembled unit was taken to Nair Hospital, Mumbai and checked on BPL machine, also it is checked on Holter cardiography, MARQUETTE, for 20 minutes recording, the keypress is changed every after 1 minute. The results obtained are satisfactory. The scripts are attached. Refer Figures 4.14,4.15,4.16,4.17,4.18, 4.19, 4.20, 4.21. Also a Holter cardiography report is attached. Attached herewith with its own analysis. This is resembling to the ECG/arrhythmia simulation. Thus a portable unit is developed as shown in figure 4.22 which able to carry at any CCU checking proper functioning of any ECG/arrhythmia monitor.

4.6.1 HOLTER CARDIOGRAPHY REPORT

HOLTER REPORT

Patient : xxx

DEPARTMENT OF CARDIOLOGY
B Y L NAIR HOSPITAL & T N MEDICAL COLLEGE
DR. A L NAIR ROAD, BOMBAY - 400 008
PH: HOSP (022) 3081490, ICCU - 3081758

ID :
Age :
Sex :

Date : 01-JUL
Hook-up date : 14-MAR
time : 08:00:
Duration : 00:30:

Referred by :
from :

Indications :
Medications :

SUMMARY

1738	QRS complexes		
251	Ventricular ectopics	which represent	14 % of total QRS complexes
1120	Supraventricular ectopics	which represent	65 % of total QRS complexes
	Paced QRS complexes	which represent	% of total QRS complexes

VENTRICULAR ECTOPY

49	Isolated			
0	Bigeminal Cycles			
1	Couplets			
4	Runs			
200	Beats in Runs			
191	Beats LONGEST at	218 BPM at 08:26:09	14-MAR	
191	Beats FASTEST at	218 BPM at 08:26:09	14-MAR	

HEART RATES

25	MIN at 08:25:01	14-MAR	
47	AVG		
133	MAX at 08:12:13	14-MAR	

SUPRAVENTRICULAR ECTOPY

120	Isolated			
25	Couplets			
27	Runs			
950	Beats in Runs			
520	Beats LONGEST at	271 BPM at 08:16:01		14-M
4	Beats FASTEST at	271 BPM at 08:12:24		14-M

S-T LEVELS Channel
mm at
mm at

LONGEST RR at

INTERPRETATION :

This recording shows the following
1] Atrial bigeminy
2] Sinus tachycardia
3] Sius bradycardia
4]Ventricular bigeminy
5] Paced beats
6] Sinus pauses
7] Asystole
8] Ventricular trigeminy
9] Ventricular trigeminy[2]
10] Runs [VPB]
11] Ventricular firillation
12] Ventricular tachycardia

Signed : Dr. N. Vijaykumar

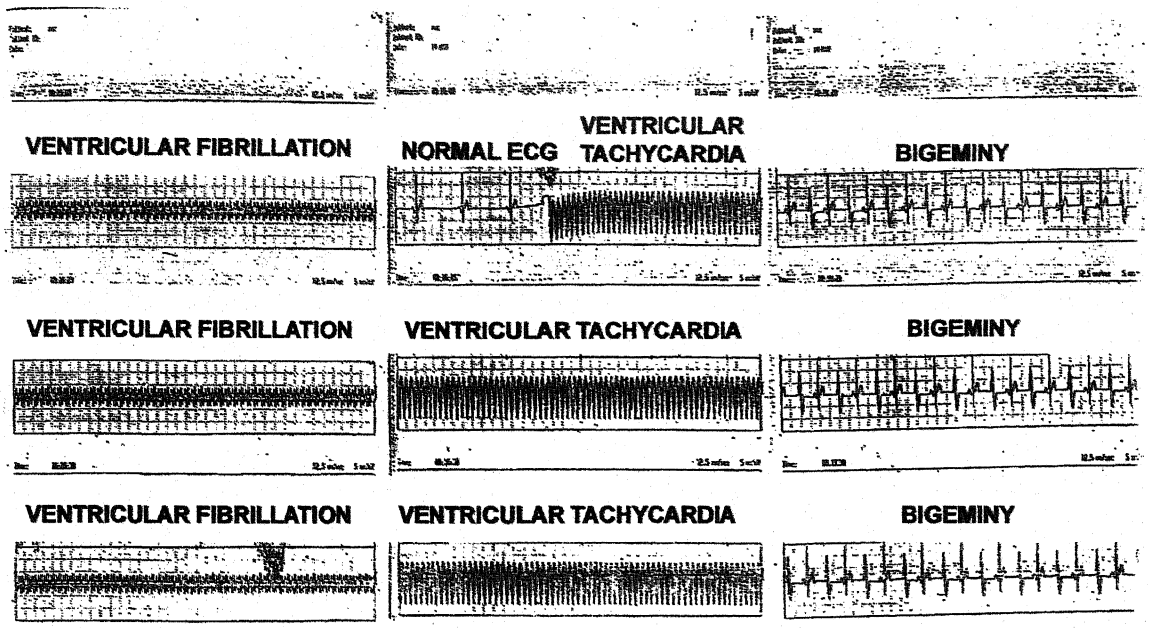


Fig. 4.14 : Arrhythmia simulation report 1 obtained at Nair Hospital, Mumbai on Holter cardiography

Patient : xxx
Patient ID:
Date : 14-Mar-06

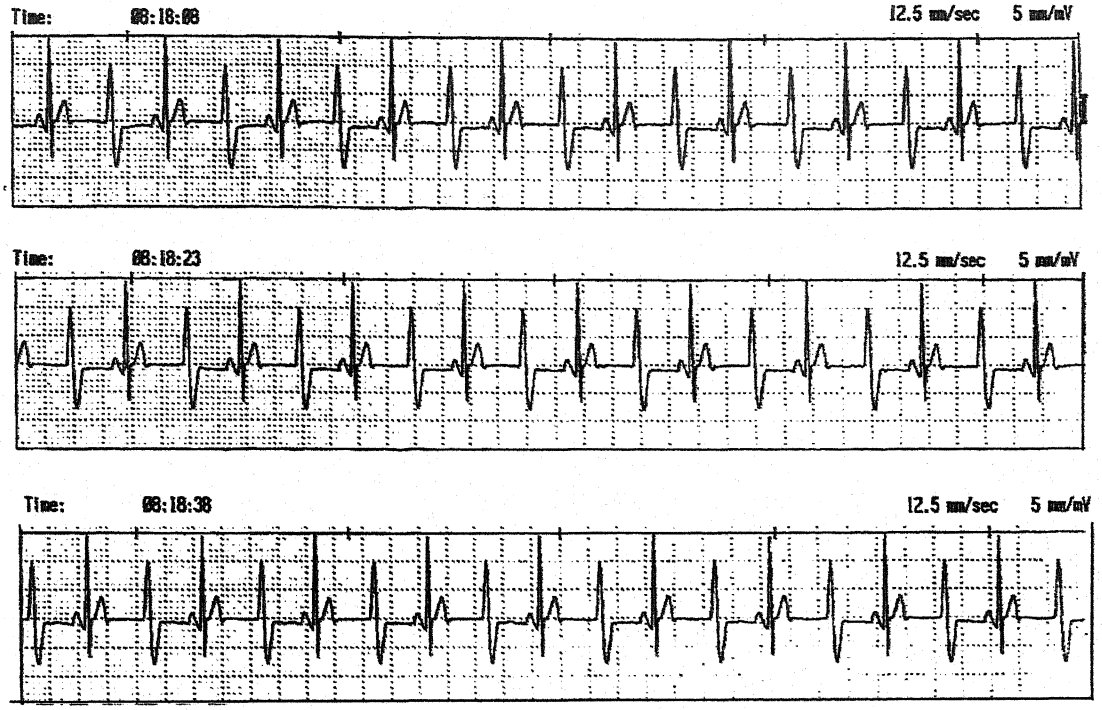


Fig. 4.15 : Arrhythmia simulation report 2 obtained at Nair Hospital, Mumbai on Holter cardiography

Patient : xxx
Patient ID:
Date : 14-Mar-06

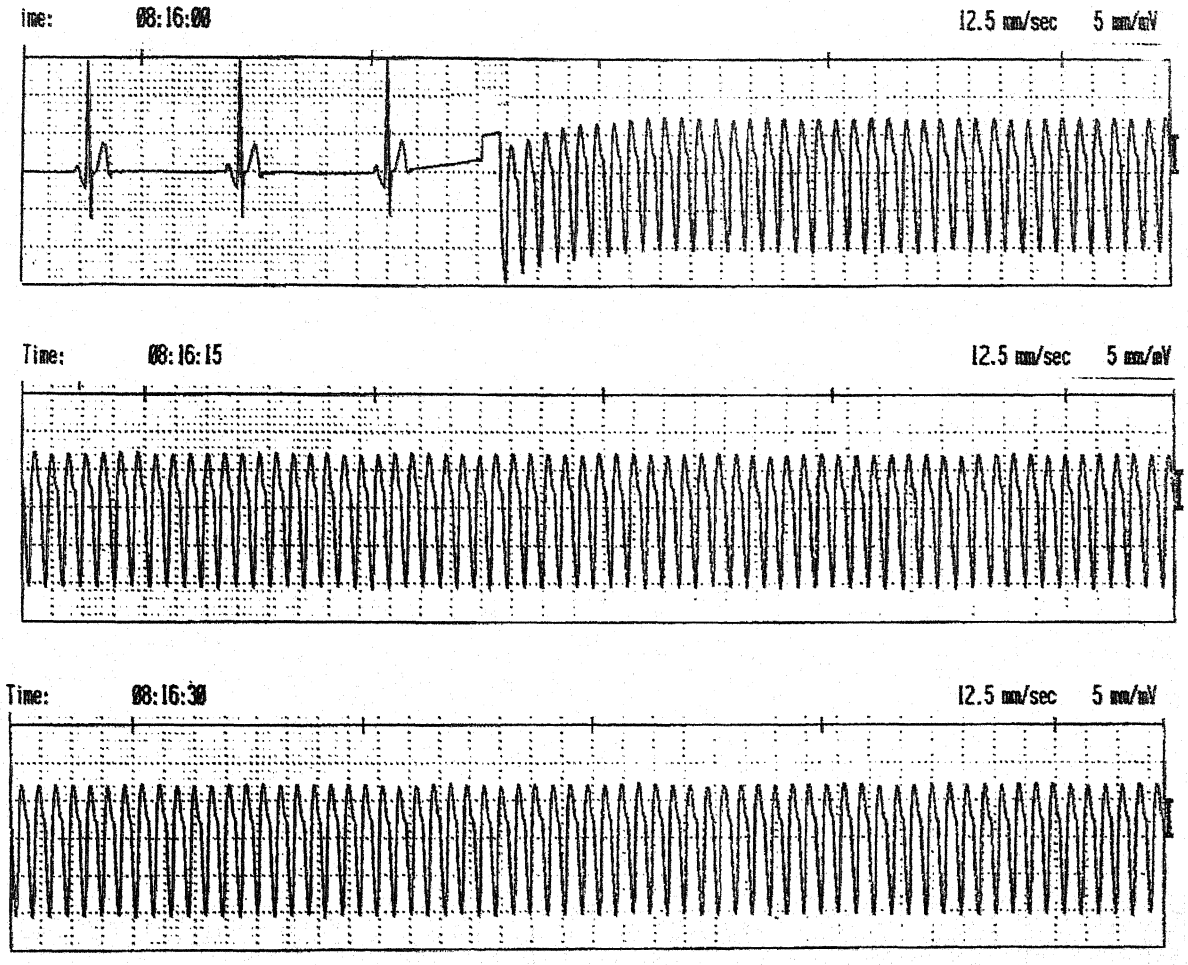


Fig. 4.16 : Arrhythmia simulation report 3 obtained at Nair Hospital, Mumbai on Holter cardiography

Patient : xxx
Patient ID:
Date : 14-Mar-06

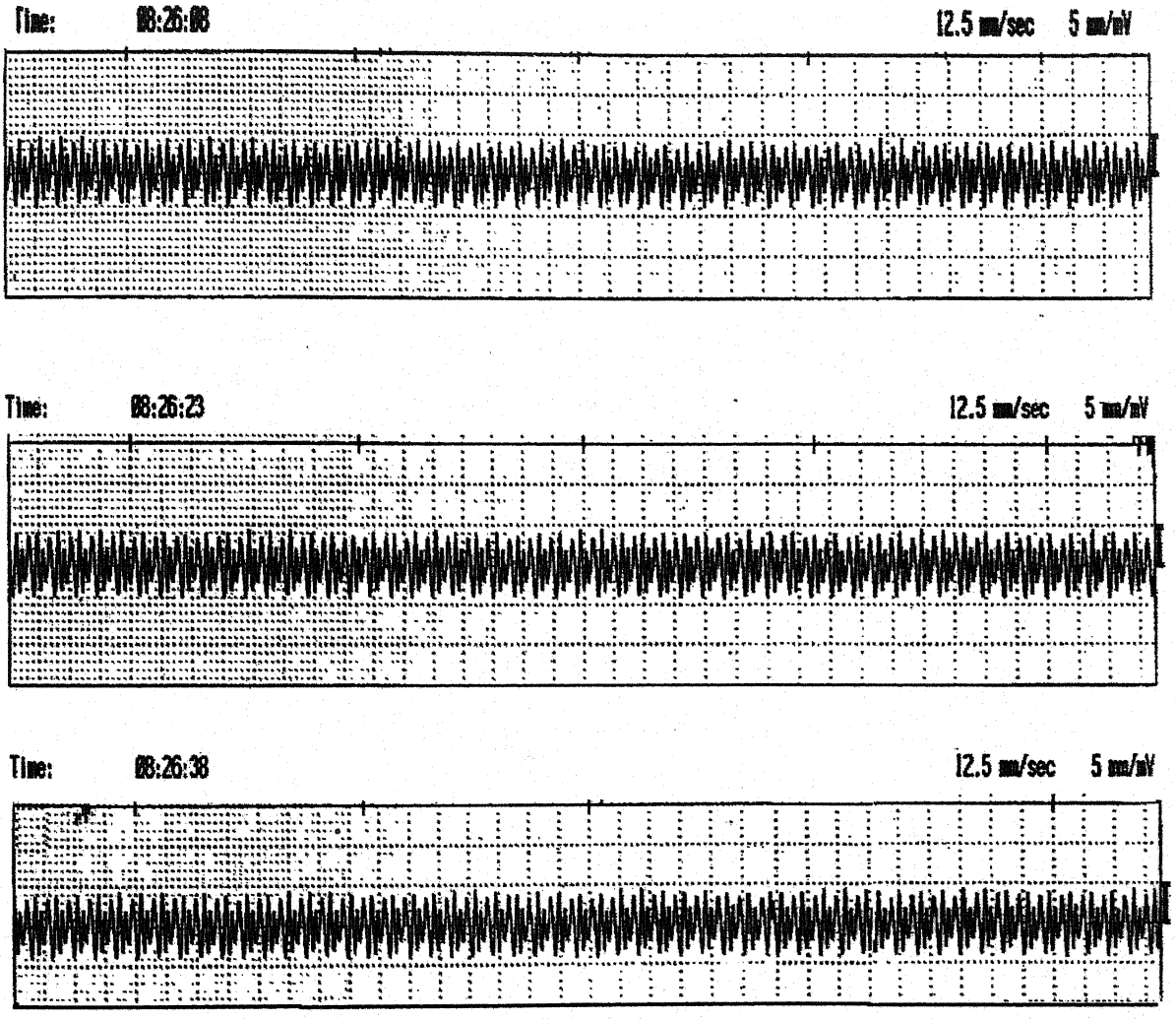
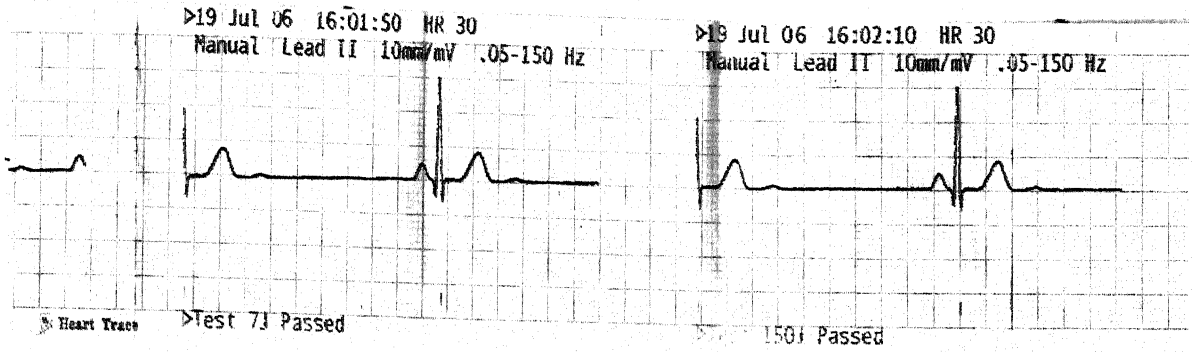
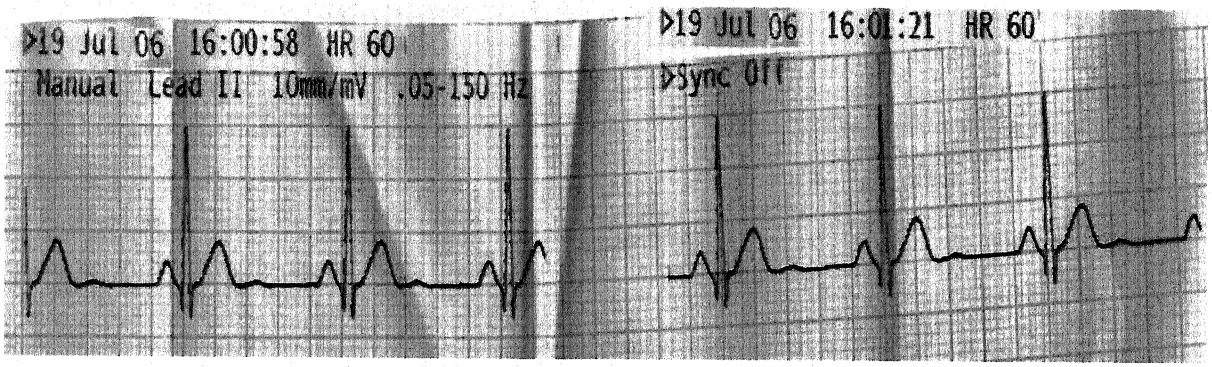


Fig. 4.17 : Arrhythmia simulation report 4 obtained at Nair Hospital, Mumbai on Holter cardiography





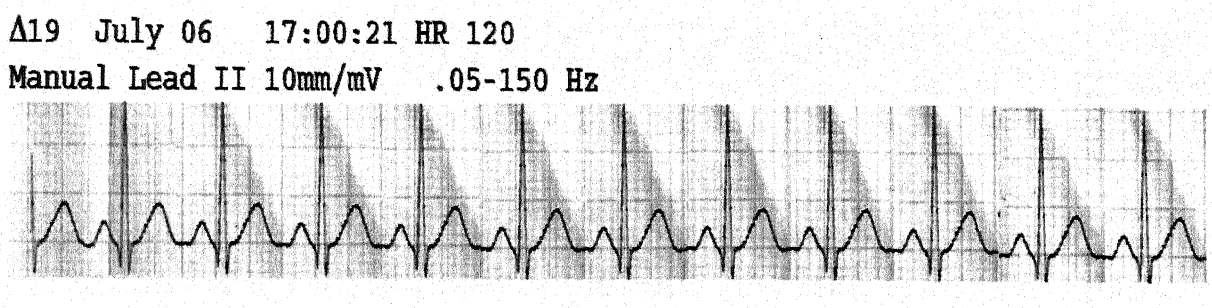


Fig. 4.18 : Arrhythmia simulation report 5 obtained at from cardiomin monitor.

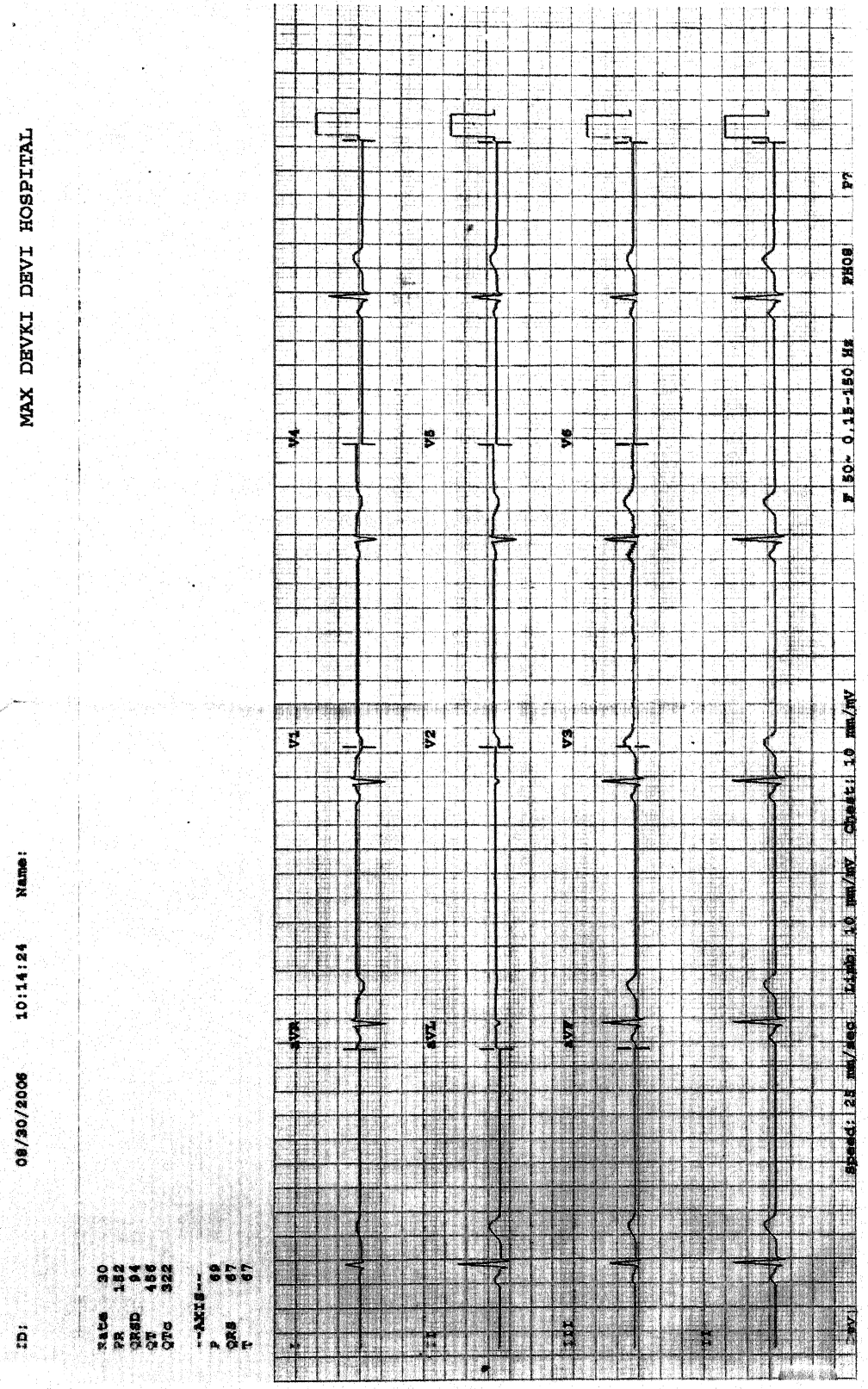


Fig. 4.19 : Arrhythmia simulation report 6 obtained at from cardiac monitor.

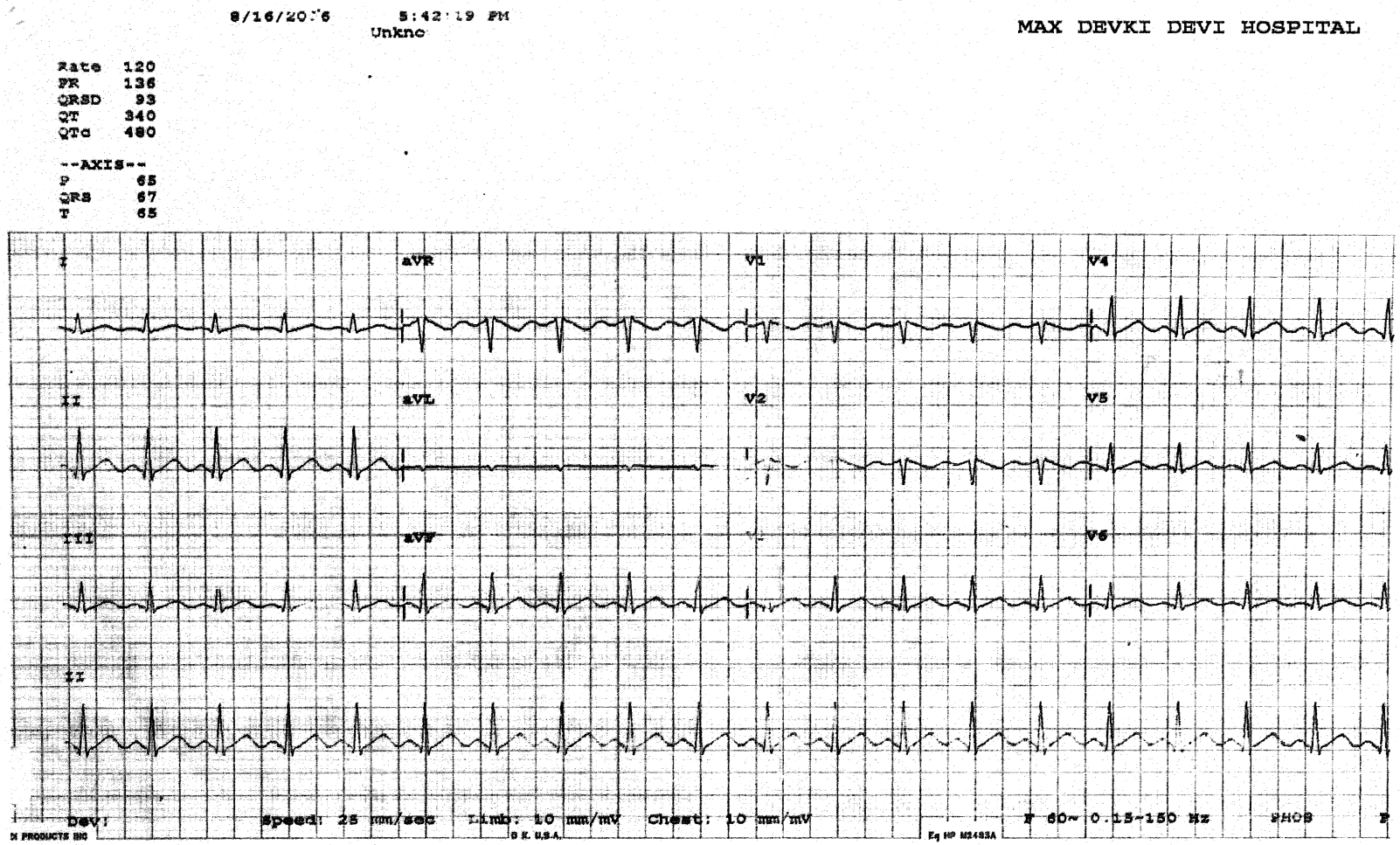


Fig. 4.20 : Arrhythmia simulation report 7 obtained at from cardiac monitor.

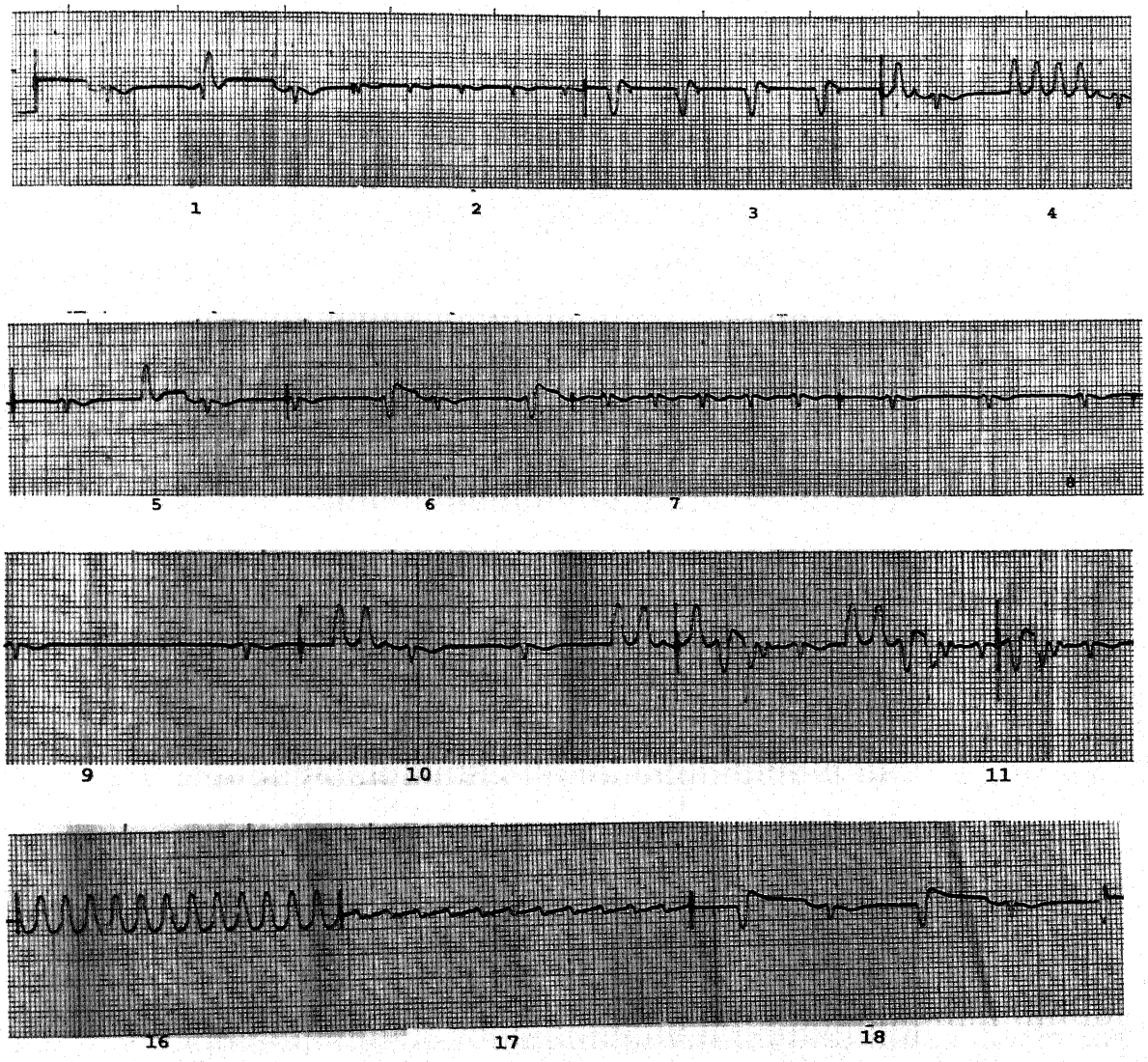


Fig. 4.21 : ECG / Arrhythmia simulation report 8 obtained on BPL 108T ECG Machine (Lead V1 Waveforms of normal ECG and 17 Arrhythmias)

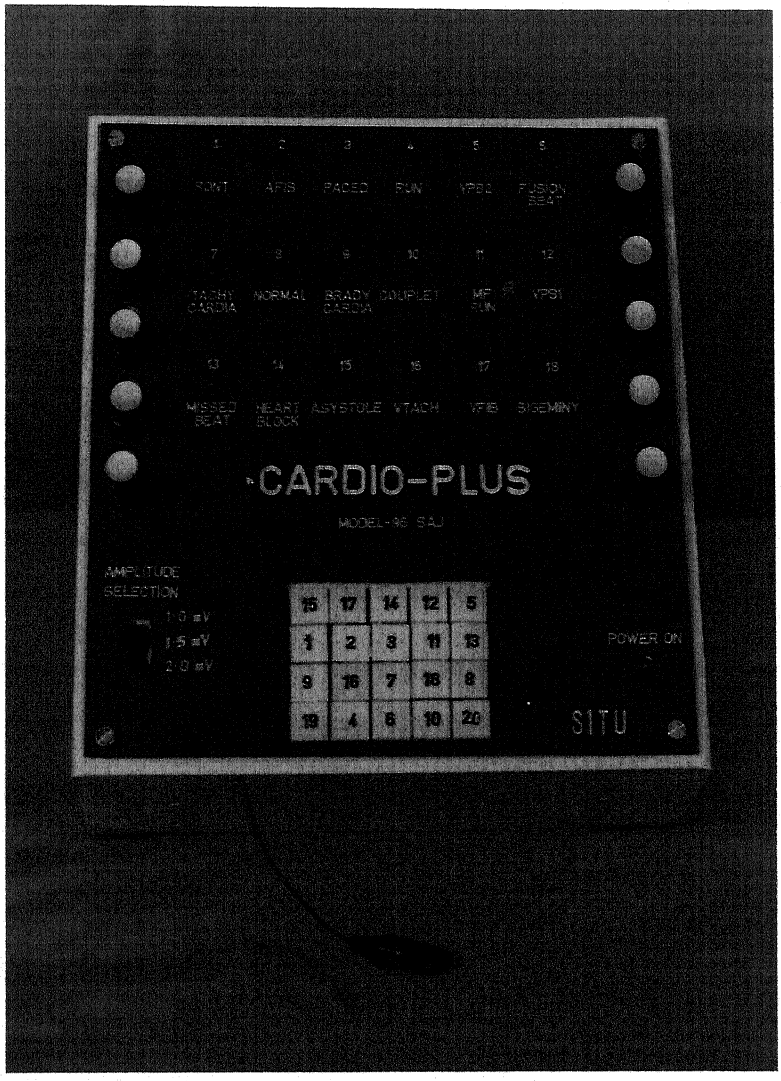


Fig. 4.22 : ECG / Arrhythmia simulator using microcontroller

CHAPTER – V

ECGSIM PROGRAM FOR QRS SIMULATION

5.1 INTRODUCTION

ECGSIM is an interactive simulation program [95] that enables to study the relationship between the electric sources of the ventricular myocardium and the resulting potentials on the thorax (QRST waveforms as well as body surface potential maps) [96] and on the heart surface (electrograms, potential maps, and maps of the local transmembrane potential). Both the depolarization [103,104,105] and the repolarization phase are covered, but not the electric activity of the atria.

One of the objectives for ECGSIM is to serve as a research tool for those interested in testing any hypothesis they may have regarding the manifestation of cardiac malfunctioning in the electrocardiographic waveforms on the thorax [105].

Another objective is for it to serve as an educational tool, to be used for students learning the basic aspects of the genesis of the electrocardiogram.

ECGSIM is not a diagnostic tool. Any diagnostic application is only indirect; ECGSIM provides a forward simulation and does not solve the inverse problem.

5.2 ECGSIM CONTENTS

1. Introduction
2. Basic Usage
3. File
4. Heart
5. Thorax
6. Membrane
7. ECGs
8. Movie
9. Help
10. Alternative Geometry

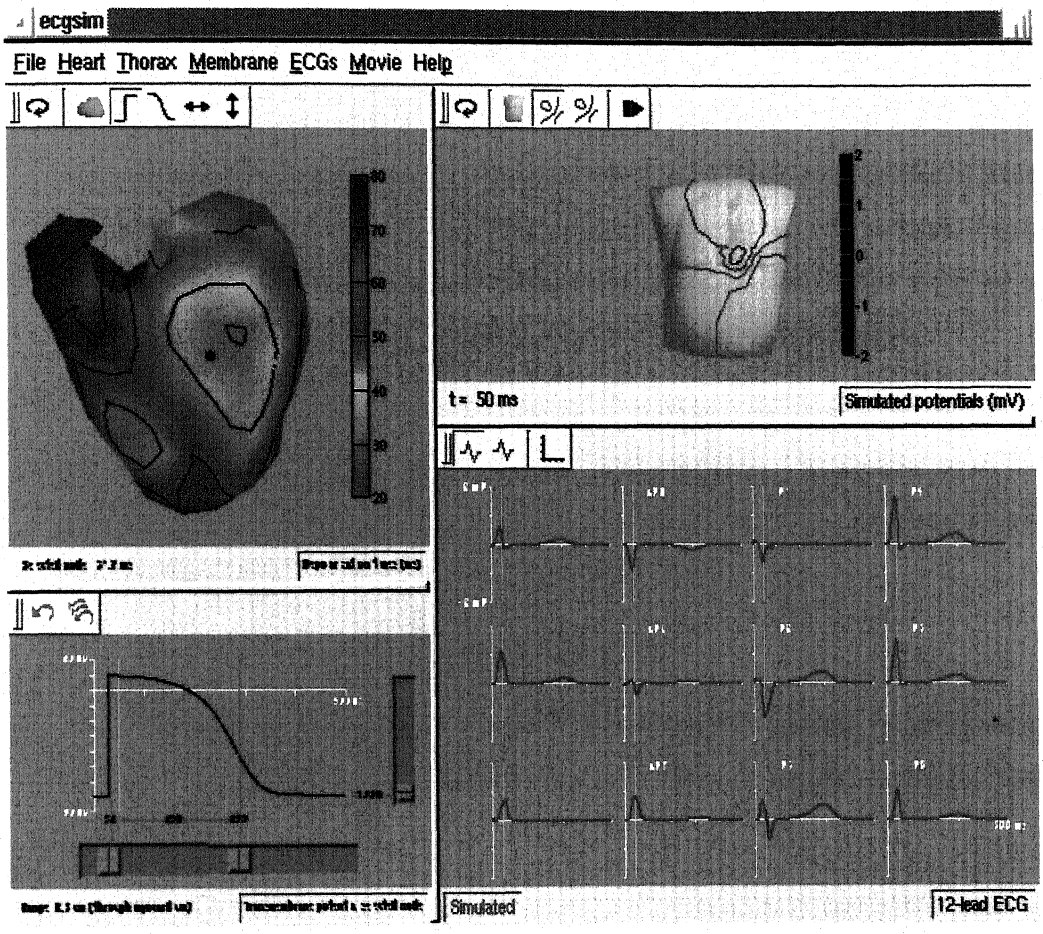


Fig. 5.1 : ECGSIM Window

5.3 ECGSIM ALGORITHMS

ECGSIM simulates electrocardiographic potentials [106] by specifying the distribution of the timing of depolarization, dep(n), and repolarization, rep(n), of the transmembrane potentials at nodes n, (n=1...N), where N is the number of nodes at the surface bounding the ventricles, referred to henceforth as the heart surface. For every node the difference apd(n)=rep(n)-dep(n) is taken a measure of the local action potential duration. The ventricular surface is the closed surface bounding all ventricular mass, i.e.,

endocardium, epicardium and their connection at the base of the ventricles. The magnitude of the upstroke of the local transmembrane potential, str(n), representing the local source strength, is allowed to be non-uniform. By setting pre-computed values for the parameters dep(n) and rep(n) for each node and using a uniform str(n) values, potentials are simulated that closely resemble the set of reference values recorded on a healthy subject.

The program allows the interactive changing of the local timing of depolarization and repolarization as well as the magnitude of the upstroke of the local transmembrane potentials. The former two sets of parameters allows to study the effect of changes in local timing, the latter set, the manifestation of local ischaemic regions.

The strength of the cardiac equivalent generator involved is the distribution on the heart surface [24,26] of the local transmembrane potential. The transfer used to determine the expression of these sources as body surface potentials was computed by applying the laws of current flow to an inhomogeneous torso model.

5.4 BASIC USAGE OF ECGSIM WINDOW

The interactive operation of ECGSIM is almost entirely mouse controlled. The main mouse operations are : dragging (moving the mouse while holding the mouse button down) and clicking of either the left- or the right mouse button.

The ECGSIM main window consists of four panes: the heart pane, the thorax pane, the membrane pane and the ECGs pane. Refer figure 5.1 for ECGSIM window. The boundaries between the planes may be shifted by using the left mouse button (drag). For each pane there is a menu item on the menu bar. The options in each menu control is displayed in the corresponding pane.

The most frequently used functions are also available from the tool buttons at the top of each pane. To view the function of each button, rest the mouse cursor on the button without pressing any of the mouse buttons. A short text explaining the function of the button will appear.

The heart and the thorax can be rotated by left mouse button dragging or by rotating the mouse wheel. The default, frontal, view is restored by clicking on the reset orientation button on the tool bar.

By clicking with the right mouse button on the heart surface in the heart plane, the nearest of the N nodes will be selected. The membrane pane shows the transmembrane potential at this node. By means of the sliders (left mouse button drag) in this pane, the parameters: onset (dep), duration (dur) and source magnitude (str) at that node and a region around it can be changed. The resulting changes are visible in the heart pane if the corresponding surface function has been selected.

The effect of these changes on the body surface potentials may be viewed in the ECGs pane and the thorax pane.

The status bar at the bottom of each pane shows properties of what is being displayed in that pane.

5.4.1 FILE

The File menu has the following entries:

- Open;
- Save;
- Default;
- Print;
- Exit.

Open

The Open menu item starts up a sub-menu containing two entries: Source parameters and Reference surface potentials..

Open : Source parameters

The parameters ECGSIM works with are: depolarization times, dep, repolarization times, rep, and the magnitudes of the local transmembrane potential, str, at all nodes of the heart. Open -> Source parameters allows the user to replace the default parameter by those read from a file. A file saved during a previous ECGSIM session, can be opened or a new can be created.

If the source parameters loaded from a file contain depolarization times beyond 500 ms, the maximum repolarisation time is automatically set to 1000 ms.

Open: Reference surface potentials

Open -> Reference surface potentials allows to replace the default set of reference potentials. These will be used both for the reference body surface potentials in the thorax pane and the reference 12-lead ECG in the ECGs pane. A file saved during a previous ECGSIM session, can be opened or a new can be created.

The number of rows in the file must match the number of nodes on the thorax. Reference surface potentials may also be loaded when ECGSIM starts by using the- ref command line option.

Save

The Save menu item starts up a sub-menu containing three entries:, Source parameters, simulated surface potentials, and simulated 12 lead ECG.

Save: Source parameters

The set of depolarization times, dep, repolarization times, rep, and transmembrane potential magnitudes (str) at all nodes of the heart can be saved to a file by using Save -> Source parameters..

Save: Simulated surface potentials

Save -> Simulated surface potentials allows the user to save the simulated surface potentials at all nodes of the thorax to a file. These may later be opened to serve as reference potentials, or used for further analysis outside ECGSIM.

Save: Simulated 12-lead ECG

Save -> Simulated 12-lead ECG allows the user to save the simulated 12-lead ECG to a file. These may then be saved for further analysis outside ECGSIM.

Default

Once a source description file is opened , the default set of source parameters is lost. You may restore the default source parameters by choosing Default can be restored.

Print

The Print menu item starts up a sub-menu, from which it can be chosen which pane is to be print.

ECGSIM sends a bitmap copy of the pane to the printer, hence the resolution of the print-out is that of the screen and not that of the printer (which commonly has a much higher resolution).

5.4.2 FILE FORMATS

File format explain the source description files, surface potential files and 12 lead ECGs file

Source description files

The default extension of the files in which ECGSIM stores the source parameters is .src. The format of the file is:

N 3

d(1) r(1) s(1)

d(2) r(2) s(2)

. . .

. . .

d(N) r(N) s(N)

where N is the number of rows that follow (=the number of nodes at the heart; N=257 for the default heart geometry), 3 is the number of columns, d(n) is the depolarization time at node n in ms, r(n) is the repolarization time at node n in ms, and s(n) is the depolarization magnitude at node n. The scaling of the latter ranges from a value of 0 for inactive nodes to 1 for the full (relative) magnitude of the upstroke of the transmembrane potential.

The index n of a selected heart node may be displayed on the status bar of the heart pane by displaying the geometry in that pane and selecting the node.

Surface potential files

The default extension of the files in which ECGSIM stores surface potentials is .bsm.

The format of the file is *the standard format used for matrix files* in ECGSIM:

L

T

p(1,1) p(1,2) ... p(1,T)

p(2,1) p(2,2) ... p(2,T)

. . . .

. . . .

p(L,1) p(L,2) ... p(L,T)

where L is the number of rows that follow (=the number of nodes on the thorax; L=300 for the default torso geometry), T is the number of columns (=number of time samples) and p(l,t) is the potential in mV at node l at sampling time t.

The sample frequency is 1000 Hz

The index of the selected thorax node may be displayed on the status bar of the thorax pane by displaying the geometry in that pane and selecting the node.

12-lead ECG files

The default extension of the files in which ECGSIM stores 12-lead ECGs is .ecg. The format of the file is:

L T

p(1,1) p(1,2) ... p(1,T)

p(2,1) p(2,2) ... p(2,T)

. . . .

. . . .

p(L,1) p(L,2) ... p(L,T)

where L is the number of rows that follow (=the number of leads, normally 12), T is the number of columns (=number of time samples) and p(l,t) is the potential in mV at lead l at time sample t.

Rows 1-6 correspond to leads V1-V6, rows 7-9 to leads aVR, aVL and aVF, respectively and rows 10-12 to leads I, II and III, respectively.

The implied sample frequency is 1000 Hz.

Triangulated geometry files

The format of the files that describe triangulated geometries is as follows:

npn
t

1 x(1) y(1) z(1)

2 x(2) y(2) z(2)

. . . .

. . . .

npnt x(npnt) y(npnt) z(npnt)

ntri

1 ind(1,1) ind(1,2) ind(1,3)

2 ind(2,1) ind(2,2) ind(2,3)

. . . .

. . . .

ntri ind(ntri,1) ind(ntri,2) ind(ntri,3)

where npnt is the number of vertices, x(i), y(i) and z(i) are the coordinates (in meters) of vertex i, ntri is the number of triangles, and ind (j,1), ind(j,2) and ind(j,3) are the indices of the vertices of triangle j. The order of the indices for a triangle defines the orientation of the triangle; when viewed from the outside the vertices are numbered clockwise.

5.5 HEART PANE

The heart pane is used to display the geometry of the heart and any of a wide range of functions on its surface. If a node on the heart is selected by right a mouse button click, its position is shown by a black patch. Refer Figure 4.24 for the Heart Pane.

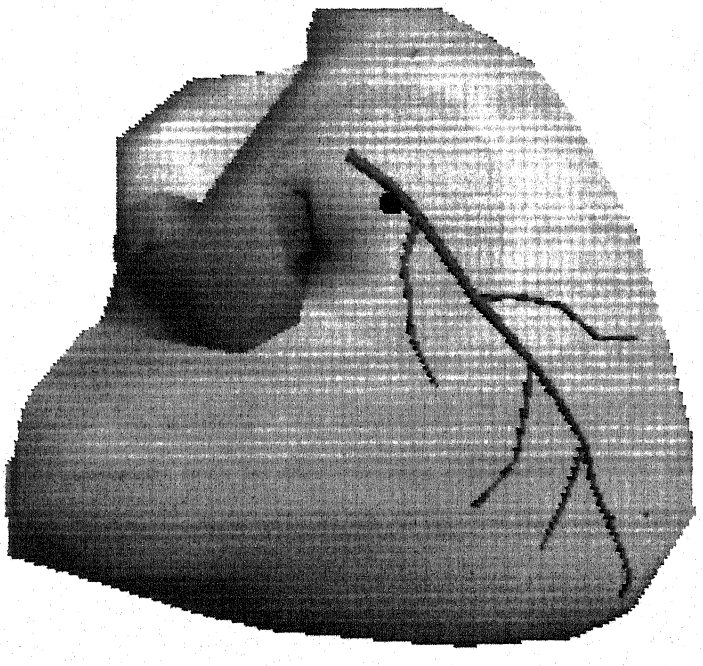


Fig. 5.2 : The Heart Pane

The function that is being displayed in the heart pane, as well as various display options that can be selected from the Heart entry on the menu bar:

- Surface function;

- Display options;
- Scale;
- Orientation.

Mouse actions in the heart pane are reflected in this pane and may also have an effect on the other ones.

The image shown in the heart pane may be copied to the clipboard by selecting Copy from the Heart menu, or by pressing Ctrl-C while the mouse is within the heart pane.

5.5.1 SURFACE FUNCTION

The item Surface function in the heart menu brings up a dialog window, with which you may choose the function to be displayed on the heart surface. Options available are: the depolarization times, repolarization times, action potential duration, action potential magnitude, transmembrane potential, epicardial potential and the transfer function.

The most frequently used surface functions may also be activated by clicking on the corresponding button on the tool bar at the top of the heart pane.

If transmembrane or epicardial potentials are displayed, the status bar indicates the time instant for which the potentials are displayed. The up and down arrow keys can be pressed to in- and decrease time by steps of one millisecond. Use the page up and page down keys for in- and decrements with steps of ten milliseconds. It can be clicked in the membrane and ECGs pane on the time instant for which the potentials are required to be displayed. Finally, the movie option is used to view the development in time of the potentials as a movie.

5.5.2 DISPLAY OPTIONS

The item Display options in the Heart menu brings up a dialog window in which it is chosen how the heart and the surface function are to be displayed. There are two tabs: the Geometry tab that controls the geometry options, and the Surface function tab that controls the surface function options.

Display options: Geometry

To facilitate orientation, the left arteria decendens marking the boundary between the right and left ventricle may be brought into view. In addition, the precordial thoracic electrodes may be included in the display. In the geometry tab, it can be selected whether the electrodes to be shown, and whether the electrodes should be transparent or opaque.

It can be chosen whether the size of the range around the selected node that is affected by changes of the source parameters at the selected node should be shown on the heart surface. It can be chosen to display the outline of the range, to dim the region of the heart surface that is within the range, or to dim the region outside the range. The heart surface may be hidden or made transparent. If a function is mapped on the heart surface using a color code, the surface will always be opaque, since a colored transparent heart is too confusing. To map a function on the heart while being able to Refer through the surface, use isofunction lines only.

The heart vector and vector loop (according to the Frank leads) for the simulated data may be displayed by choosing opaque for the corresponding items. The scale of the vector is 13 cm/mV times the scale factor of the signals in theECGs pane. The size of the displayed vector can be changed by using the scale in the ECGs pane.

Display options: Surface function

The surface function tab allows to determine how the function is displayed; by a color map, isofunction lines, or by both. Also, it is determined whether the light effect should be active when using a color map display. The light effect gives a three-dimensional impression of the geometry of the heart, but it also renders the colors on the surface less bright.

5.5.3 SCALE

The item Scale in the heart menu brings up a dialog window, through which the scale can be set for the various surface functions that are plotted on the heart surface.

By default, the scale of time functions (repolarization, depolarization and action potential duration) is adapted automatically as the timing values change. By unchecking the Autoscale check box, manual control by means of the three timing sliders is enabled.

5.5.4 MOUSE ACTIONS IN THE HEART PANE

Orientation

The heart can be rotated by (left) mouse dragging. By rotating the mouse wheel, the heart rotates along an axis perpendicular to the screen. If the shift key is pressed simultaneously, the angle by which the heart rotates with each click of the mouse wheel becomes smaller.

Selection

A node at the heart is selected by a right mouse button click on the heart surface. The node nearest to the "click" is selected, as shown by a black patch on the surface. Only one node may be selected at a time; if another node is selected, the previous one is deselected. If the right mouse button is clicked outside the heart, the currently selected node is deselected.

If a function is mapped on the heart surface, the value at the selected node is displayed on the status bar of the heart pane. If the geometry is displayed, the location of the selected node and its index are displayed (the node index corresponds to the row in which the source parameters for that node aresaved).

In the membrane pane the transmembrane potential of the selected node is displayed.

Setting the range

The size of the range around the selected node that is affected by changes of the source parameters at the selected node may be set by clicking the middle mouse button. The point that was clicked on will define the edge of the selected range.

The size of the range may also be set from the range dialog window in the membrane menu. In this dialog window it may also be chosen whether the range should be computed through the myocardium (transmuraly), or merely on the part of the heart surface that carries the selected node.

5.6 THORAX PANE

The thorax pane is used to display the geometry of the thorax, and any of a wide range of functions on the thorax surface. If a node on the thorax is selected, it is shown by a black patch on the surface.

What exactly is displayed in the thorax pane and how, is controlled by the various menu options available from the Thorax entry on the menu bar:

- Surface function;
- Display options;
- scale;
- orientation.

Mouse actions in the thorax pane are reflected in this pane and may also have an effect on the other ones.

The contents of the thorax pane is copied onto the clipboard by selecting Copy from the Thorax menu, or by pressing Ctrl-C while the mouse is within the thorax pane.

5.6.1 SURFACE FUNCTION

The item Surface function in the thorax menu brings up a dialog window, with which the function to be displayed is chosen on the thorax surface. Available are the simulated potentials (i.e. the potentials that result from the transmembrane potentials at the heart as set by the user), the reference (i.e. measured) potentials, and the transfer function.

The most frequently used surface functions may also be activated by clicking on the corresponding button on the tool bar at the top of the thorax pane.

If surface potentials are displayed in the thorax pane, the status bar indicates the time instant for which the potentials are displayed. The up and down arrow keys can be pressed to in- and decrease the time by steps of milliseconds. The page up and page down keys are used for in- and decrements with steps of ten milliseconds. Also, in the membrane or in the ECGs pane can be clicked to select the time instant for which the potentials are required to be displayed. Finally, the movie option is used to view the development of the potentials in time as a movie.

5.6.2 DISPLAY OPTIONS

The item Display options in the Thorax menu brings up a dialog window, from which it is selected how the thorax and the surface function are displayed. There are two

tabs: the Geometry tab that controls the geometry options, and the surface function tab that controls the surface function options.

Display options: Geometry

In the Geometry tab it is determined whether the heart, lungs, thorax and precordial electrodes is to be displayed, and whether it is required to be transparent or opaque.

If a function is mapped on the thorax using a color code, the surface will always remain opaque, as a colored transparent thorax is too confusing. To map a function on the thorax while being able to Refer through the surface, isofunction lines only are used.

The heart vector and vector loop (according to the Frank Leads) for the simulated data may be displayed by choosing opaque for the corresponding items. The scale of the vector is 20 cm/mV times the scale factor of the signals in theECGs pane. The size of the displayed vector is changed by changing thescale in the ECGs pane.

Also, it can be chosen whether the grid and/or the nodes that make up the discretized thorax are to be displayed.

Display options: Surface function

The surface function tab allows to determine how the function is displayed; by a color map, isofunction lines, or both. Also, it is determined whether the light effect should be active when using a color map display. The light effect gives a three-dimensional impression of the geometry of the thorax, but it also renders the colors on the surface less bright.

5.6.3 SCALE

The item Scale in the thorax menu brings up a dialog window, by which the scale for the various surface functions that can be plotted on the thorax surface is set. This dialog window may also be started by clicking on the Scale button on the thorax tool bar.

5.6.4 MOUSE ACTIONS IN THE THORAX PANE

Orientation

The thorax can be rotated by left-mouse dragging, i.e. move the mouse while holding the left mouse button down. By rotating the mouse wheel, the thorax rotates along an axis perpendicular to the screen. If the shift key is pressed simultaneously, the angle by which the thorax rotates with each click of the mouse wheel becomes smaller.

Selection

A node at the thorax is selected by clicking with the right mouse button on the thorax surface. The node nearest to the "click" is selected, as shown by a black patch on the surface. Only one node may be selected at a time. If another node is selected, the previous one is deselected. If the right mouse button is clicked outside the thorax, the currently selected node is deselected.

If a function is mapped on the thorax surface, the value at the selected node is displayed on the status bar of the thorax pane. If the geometry is displayed, the index of the selected node is displayed (the node index corresponds to the row in which the surface potentials for that node aresaved).

In the ECGs pane, it can be chosen to display just the (single) ECG at the selected node.

5.7 MEMBRANE PANE

The membrane pane displays the transmembrane potential at the selected node of the heart surface. If desired, the electrogram at that node may also be displayed. Refer Figure 5.3 for Membrane Pane.

The sliders allow to change the parameters describing the transmembrane potential: the depolarization time (the fast onset of the action potential), the repolarization time (defined as the moment of maximum down-slope during repolarization), and the magnitude of the action potential. The action potential duration that results is taken to be the difference between the specified repolarization and depolarization times.

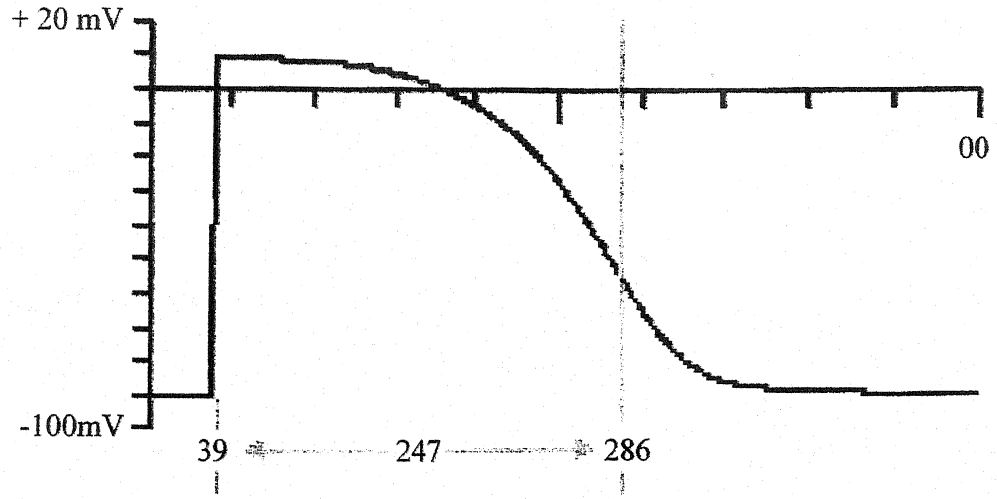


Fig. 5.3 : The Membrane Pane

The left knob of the slider pair beneath the plot allows to specify the onset (dep) of the action potential at the selected node. The right knob affects the depolarization time (rep). When the left slider knob is moved the right one moves along. In this way the action potential duration (apd) remains constant. Moving the right knob sets the repolarization timing (rep) only. This also affects the duration since rep = dep+apd. The numbers shown indicate (from left to right): dep, apd and rep.

The slider on the right is used to change the action potential magnitude, expressed as a fraction of its normal value.

If the user changes the source parameters, the effect on the ECG is immediately visible in the other panes, provided that the appropriate functions have been selected.

The Membrane menu on the menu bar contains the following entries:

- undo;
- undo all;
- show electrogram;
- Focus;
- Range;

- Maximum repolarisation time;
- statistics;
- Repolarisation waveform.

The image shown in the membrane pane may be copied to the clipboard by selecting Copy from the Membrane menu, or by pressing Ctrl-C while the mouse is within the membrane pane.

FOCUS

If Focus from the Membrane menu is chosen, the currently selected node on the heart surface will be a focus of depolarization. A dialog window will appear that allows to control the parameters (e.g. propagation velocity) of the depolarization wave front starting from this focus.

In this dialog window, the depolarization time of the focus can be set, and the speed of the depolarization wave front started from this focus. Furthermore it is chosen whether the selected node should be the only focus of depolarization (Replace current activation sequence), or whether the node should be an additional focus (Add to current activation sequence). In the latter case the original depolarization times will be retained for nodes that are reached by the new depolarization wave front at a later time than its original depolarization time.

If a focus of depolarization is defined on the heart surface, the repolarization time rep(n) of each node n is set as follows:

$$rep(n) = dep(n) + OldDur(n) + 0.4\,(OldDep(n)-OldMeanDep) - 0.4\,(dep(n)-MeanDep),$$

where OldDur(n) is the original action potential duration of node n (i.e: before the focus was defined), OldDep(n) is the original depolarization time of node n, and MeanDep and OldMeanDep are, respectively, the new and original mean depolarization times.

This, heuristic, assignment of repolarization times is based on the following arguments. The original distribution of the action potential duration is partially an expression of the intrinsic characteristics of the myocardium. For another part it is the result of the distribution of the activation times, where regions that depolarize early tend to repolarize late. Hence the new action potential duration is set the original one, plus a

term that is negative is the new activation time is later than before, and vise versa. The weight factor 0.4 is based on the statistical relation between depolarization and repolarization times.

RANGE

The parameter values of nodes within a certain range around the selected node are also adapted. The relative amount by which these node values change changes from one at the selected node to zero at the edge of the range.

The size of the range is controlled by the range dialog window, which may be activated from the membrane menu. In this dialog window it is also chosen whether it is required to compute the range through the myocardium, or along the myocardial surface. In this way various myocardial malfunctions may be modeled. For instance, a bundle branch block would affect both epicardium and endocardium in roughly the same way. So here the range is chosen through the myocardium. On the other hand, hypertrophy, may be modeled by increasing the activation times at the epicardium only. In such cases it is desired to restrict the range to the epicardial surface.

As an alternative, the size of the range is also set by clicking with the middle mouse button on the heart surface displayed in the heart pane . If some parameter(s) of the selected node are changed, and subsequently the range is changed, the changes will be recomputed for the new range. If it is required to make new changes on top of the old ones for a different range, first select another node and then come back to this node.

UNDO

Recent changes may be undone by selecting Undo from the Membrane menu, or by clicking on the Undo tool button on the tool bar at the top of the membrane pane.

By selecting Undo All, or clicking on the Undo All tool button, all changes made since the start of the program or since the last time a source parameters file was opened are undone. Once a source parameter file is opened, one can go back to the default source parameters (i.e. the ones with which the program starts) by selecting Default from the File menu.

SHOW ELECTROGRAM

This option is used in the membrane menu to show and hide the electrogram at the selected node on the heart surface.

MAXIMUM REPOLARIZATION TIME

By default, the maximum value for the repolarization time of any node is 500 ms. This is adequate for most cases, but in some cases, e.g. the simulation of the long QT syndrome, later depolarization times are needed.

By selecting Maximum repolarization time from the Membrane menu, a dialog window is opened that allows to choose between the values 500 ms and 1000 ms for the maximum repolarization time. All sliders involving depolarization time or action potential duration are adapted to the selected maximum repolarization time.

When reducing the maximum repolarization time from 1000 ms to 500 ms, there may be nodes that have a repolarization time beyond the new maximum. If such is the case, the user is prompted to consider whether these values should be clipped to 500 ms.

If a source parameter set loaded from file contains repolarization times beyond 500 ms, the maximum repolarization time is automatically set to 1000 ms.

STATISTICS

By selecting the Statistics item from the Membrane menu, a dialog window is opened that displays information about some basic statistics of the timing parameters. In addition it allows the user to scale these statistics. This is effected by merely scaling and/or shifting the involved source parameters, while keeping the pattern of their distribution in tact.

The dialog window contains a table that displays the mean, standard deviation, minimum, and maximum of the depolarization times, repolarization times, and action potential durations.

The dialog window also contains sliders to scale the means and dispersions (standard deviations) of these timing parameters. The result of these changes are

immediately visible in the various panes. Wile this dialog window is being displayed, all controls outside the dialog window are inactive.

The size of the sliders and slider knobs are automatically adjusted to ensure that no non-valid values of the timing parameters are generated (i.e. depolarization before 0 ms, repolarization beyond 500 ms, or action potential duration less than 50 ms). Also, scaling of the depolarization dispersion will lead to changes in the action potential duration dispersion and vice versa.

REPOLARIZATION WAVEFORM

When ECGSIM starts, the overall waveform of the repolarization is computed by integrating and averaging the T-wave of the reference ECG, as described in, Genesis of the t wave based on an Equivalent surface maodel listed in the reference section. Subsequently, this waveform is adapted to fit the depolarization and repolarization time of each node at the heart.

By selecting the Repolarization waveform item from the Membrane menu, the overall waveform is recomputed from the dominant T wave estimated from the current reference ECG. Furthermore, a dialog window appears containing a slider by which the steepness of the down slope may be changed, as percentage of the slope that was computed from the reference ECG.

TIME INSTANT SELECTION

If surface potentials are displayed in the heart or thorax panes, a vertical yellow line in the membrane pane indicates the time instant for which the potentials are displayed. If the left mouse button is clicked on within the membrane pane, the potentials for the corresponding time instant are displayed in the thorax pane. The up and down arrow keys are pressed to in- and decrease the time by steps of milliseconds, and the page up and page down keys for in- and decrements with steps of ten milliseconds.

5.8 ECGs PANE

The ECGs pane displays ECGs. Refer figure 5.4 for ECGs Pane.

DISPLAY PROPERTIES

The Display properties dialog window is activated from the ECGs menu. It allows to select which ECGs are required to display: the simulated ECGs (i.e. those that result

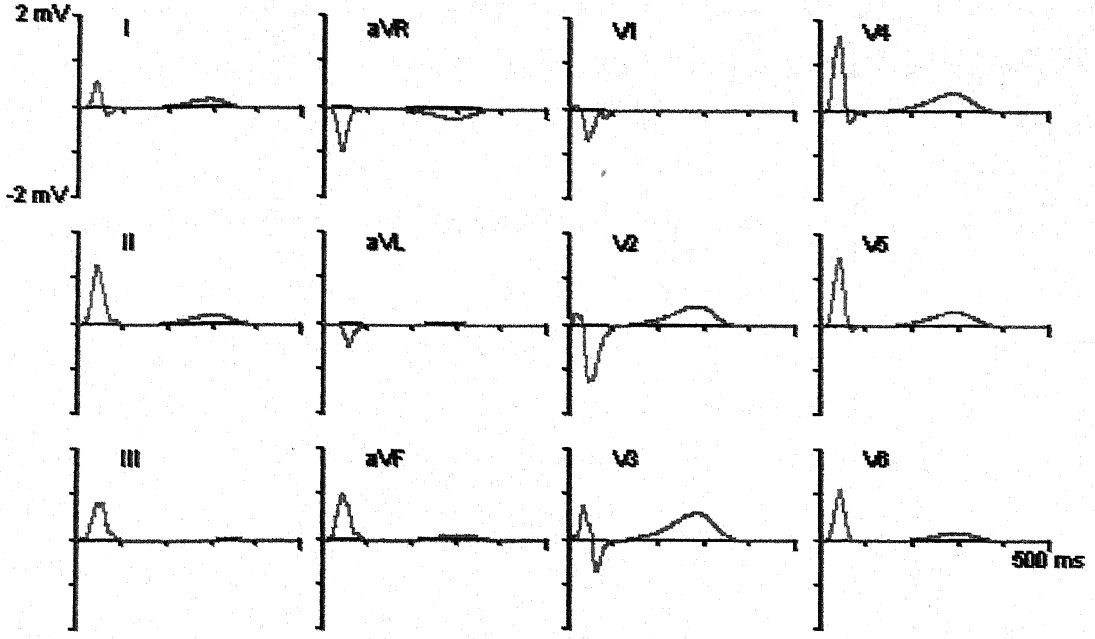


Fig. 5.4 : The ECG Pane

from the transmembrane potentials at the heart as set by the user), the reference (i.e. measured) ECGs, or both. The simulated ECGs are shown in red, the simulated ones in blue. It may be chosen which ECGs are required to display by pressing the corresponding tool buttons on the ECGs tool bar.

In the Display properties dialog window it may also be chosen whether it is desired to display the standard 12-lead ECG, the ECG at the node selected in the thorax pane, or the vectorcardiogram (VCG) according to the Frank lead system.

SCALE

In the scale dialog window, also activated from the ECGs menu, the vertical scale can be set and selected the time span required to display. The scale dialog window can be activated by pressing the scale tool button on the ECGs tool bar.

FILTERING

In the filtering dialog window, activated from the ECGs menu as well, the type of temporal filtering applied to the simulated ECGs can be chosen. This also affects the simulated ECGs in the thorax pane.

If baseline correction (the default value) is chosen, the potentials at the beginning of the P wave and at the termination of the T wave will have zero potential. This is how idealized ECGs are commonly presented. If AC filtering (the standard type of filtering in ECG recorders) is chosen, the average of the potential over time will be zero for all leads. If DC filtering is chosen, the ECGs are in fact unfiltered, i.e. the true potentials are plotted.

At the body surface, no DC recording of the ECG is possible, due to the contact potentials between the skin and the electrodes. These contact potentials slowly vary in time and have magnitudes that are generally much greater than that of the ECG. The AC-coupling that is always involved in ECG recordings effectively reduces the effect of these contact potentials. However, AC-coupling also wipes out any DC component that may be generated by the heart's electric activity. This component can never by recovered. The baseline correction that is commonly involved in the analysis of the ECG tries to overcome this problem, but is only successful if no currents are generated by the heart in the interval between successive beats.

The DC option in ECGSIM allows one to study the effect of the AC and baseline coupling of ECG amplifiers. The DC filtered (unfiltered) ECGs are Refern to differ from the baseline corrected ECGs if of regions of reduced action potential amplitude (ischemia) are present. Where in baseline corrected ECGs ischemia leads to ST elevation , the actual (DC) potentials would lead to the depression of the baseline, and vise versa. If the ST segment is much shorter than the TP interval, AC filtering results in a TP interval that is close to zero, and hence ECG recordings may look more like baseline corrected ECGs than do unfiltered ECGs.

RELATION WITH OTHER PANES

If surface potentials are displayed in the heart or thorax panes, a vertical yellow line visible in the plots indicates the time instant for which the potentials are displayed. If

the left mouse button is clicked on within the ECGs pane, the potentials for the corresponding time instant are displayed in the thorax pane. The up and down arrow keys are also pressed to in- and decrease the time by steps of milliseconds, and the page up and page down keys for in- and decrements with steps of ten milliseconds.

The temporal filtering of the simulated ECGs in the thorax pane and those in the ECGs pane are both controlled by the values set in the filtering option of the ECGs menu.

COPYING THE CONTENT OF THE ECGS PANE

The contents of the ECGs pane may be copied to the clipboard by selecting Copy from the ECGs menu, or by pressing Ctrl-C while the mouse is within the ECGs pane.

5.9 MOVIE

If surface potentials are displayed in the heart or torax panes, one can start up a movie in which the surface potentials are shown for subsequent time instances. During the movie vertical yellow lines in the membrane and ECGs panes indicate the time instant for which the potentials are displayed.

The movie can be started by selecting Start from the movie menu. It can also be started by clicking on the Start/stop movie tool button in the thorax pane.

MOVIE PROPERTIES

The movie parameters are controlled from the Movie properties dialog window, which is activated from the Movie menu.

By default, the start and end of the movie coincide with the first and last time instant shown in the ECGs pane. By unchecking the Lock to ECG span, the Start/end slider in the Movie properties is enabled, and may be used to set the span of the movie.

The speed slider controls the movie speed, which may be varied from 10 ms/s to 1000 ms/s (i.e. real time).

5.10 HELP

There are 4 ways to obtain help in ECGSIM:

1. For a comprehensive manual, choose Manual from the Help menu. This way one will start the built-in manual browser. While the manual browser is active, may

ECGSIM can be continued to operate, enabling one to experiment while consulting the manual. The manual browser is locked while a dialog window is activated.

2. If one choose What's this? from the Help menu, the mouse pointer changes into a question mark. The mouse cursor can be moved to the object within ECGSIM about which it is desired to know more, and then click on the left mouse button. A brief text with an explanation of the object's function will appear.

3. The function of the tool buttons are determined at the top of each pane by resting the mouse above them for a moment. A brief text with an explanation of the function of the button will appear.

4. The dialog windows in ECGSIM contain a button with a question mark in the title bar. If this button is pressed, the mouse cursor changes into a question mark. If one clicks on an element of the dialog window, a brief text with an explanation of the function of the element will appear.

5.11 ALTERNATIVE GEOMETRY

The default (heart and torso) geometry set built into ECGSIM can be replaced by a set constructed by anybody. Then it is required to supply the corresponding transfer function, the distance matrices used to compute along the heart surface or through the myocardium. This section of the manual describes how to incorporate alternative geometry data into ECGSIM, and the formats of the files involved.

LOADING ALTERNATIVE GEOMETRY DATA INTO ECGSIM

In order to load the alternative geometry data in ECGSIM, the file names in the command line must be specified by starting ECGSIM in the following way:

ecgsim -alt TransferFile SourceFile heartFile SurfDistFile VolDistFile ThoraxFile StdLeadsFile LungsFile

with

file	*format*	*description*

TransferFile	matrix	contains the MCG transfer matrix
SourceFile	matrix	contains initials source parameters
heartFile	geometry	contains the triangulated heart
SurfDistFile	matrix	contains the distance between nodes at the heart measured along the surface of the myocardium
VolDistFile	matrix	contains the distance between nodes at the heart measured through the myocardium
ThoraxFile	geometry	contains the triangulated torso
StdLeadsFile	Refer below	contains
LungsFile	geometry	contains the triangulated lungs (may be omitted)

When using the -alt option, use the -ref option in order to supply an appropriate reference ECG (Refer below).

STARTING ECGSIM WITH AN ALTERNATIVE REFERENCE ECG

In order to start ECGSIM with an alternative reference ECG, the file name of the reference ecg must be specified in the command line in the following way:

ecgsim -ref ReferenceFile

ReferenceFile is the name of the file containing the reference ECG. Its format is the standard matrix format (as used by ECGSIM in saving surface potentials). Each row represents the ECG as a function of time at the corresponding vertex of the thorax. Consequently, the number of rows must match the number of vertices of the thorax.

In the Windows version , the -alt command line option must be included in a short-cut by which ECGSIM is started. For that purpose, right-click on the short-cut, and add the -alt option and its parameters to the command line (after ecgsim.exe).

FORMAT OF "STANDARD LEADS" FILES

A "Standard Leads" file must have the following format:

8

1 v(1)

2 v(2)

. .

. .

8 v(8)

v(1) to v(6) indicate the index of the vertex at the thorax at which v1 to v6 are located. v(7) indicates the vertex to which VR is assigned, and v(8) the vertex to which VL is assigned (usually the tip op the right and left shoulder respectively).

aVR and aVL are constructed by multiplying the potential at v(7) and v(8) by 1.5. aVF is constructed as follows: aVF=-aVR-aVL (this assumes the transfer matrix is referenced to Wilson Central Terminal).

The potential at lead I is constructed by taking the potential at v(8) minus the one at v(7). Leads II and III are constructed in a simular fashion.

5.12 CONCLUSION

ECGSIM is an interacting simulation programme that enables to study the relationship between the electrical activity of the ventricular myocardium and the resulting potential on the thorax. It offers following features :-

- The size and position of the ECGSIM window is retained between sessions.

- The user can specify alternative heart and torso geometry;
- Activation patterns can be generated by defining foci at the epicardium;
- More Display options in the thorax pane: heart, lungs, and thorax can be hidden, made transparent or opaque.
- More Display options in the heart pane: left arteria decendens (LAD) and range of selection can be visualized.
- Transmembrane potentials and epicardial potentials can be mapped in the heart pane;.
- Electrograms can be displayed in the membrane pane.
- The heart vector and vector loop can be plotted in the heart and thorax panes;
- The slope of the repolarization phase of the transmembrane potential can be changed;
- Different temporal filtering options are available for the simulated ECGs;

CHAPTER-VI

RESULTS AND CONCLUSION

6.1 INTRODUCTION

The diagnostic ECG is usually recorded in the form of orthogonal leads, such as Frank leads or 12-lead recordings. The orthogonal lead system provides a readily interpretable picture of a cardiac vector, but the 12-lead system is now more popular as considerable clinical expertise has been acquired in interpreting the waveforms that appear in 12-lead ECGs. The instrumentation must produce high fidelity recordings followed by high-resolution digitization. Full 12-lead analysis makes certain tasks, such as reliable QRS detection, more reliable. At the same time extensive rules and algorithms are needed to classify complex arrhythmias. Interpretation based on simultaneous recordings from several leads is helpful in diagnosing conditions such as hypertrophy or myocardial infarction. The diagnostic ECG systems are useful in cardiologists offices, as portable carts in bedside recording, as patient monitors in cardiac intensive care, and so on. Modern commercial systems are based on multiple microprocessors that do sophisticated realtime ECG data acquisition and analysis. A number of monitoring systems may be connected together as in intensive care or via telephone or communication networks. They share sophisticated interpretation algorithms and large database. Diagnosis by the automated ECG interpretation system is usually not perfect and must be validated by human observers, Still ECG interpretation by computers reduce costs and makes ECG interpretation services widely available.

6.2 RESULTS

The simulation of ECG / Arrhythmia is an effective tool for monitoring the performance of the ECG / Arrhythmia monitors. The results obtained by the simulation with embedded microcontroller is tested at Nair Hospital, Mumbai on Holter cardiography and on ECG machines like BPL108T, Cardiomin at various places. The reports obtained are satisfactory and when concerned with the cardiologist, obtained the analysis as per the simulation. See the reports obtained as figures 4.14 to 4.21

6.3 COMPARISON OF THE SIMULATED REPORTS WITH THE ACTUAL PATIENTS ECG SIGNALS

The simulation results are compaired with the actual patients 12 lead ECGs. The features of the actual patients ECGs are matching with the features of the simulated ECGs as per the amplitudes, timings and morphology of the wave is concerned. Some of the comparisons are attached here.

1. AN 84 YEAR OLD LADY WITH HYPERTENSION

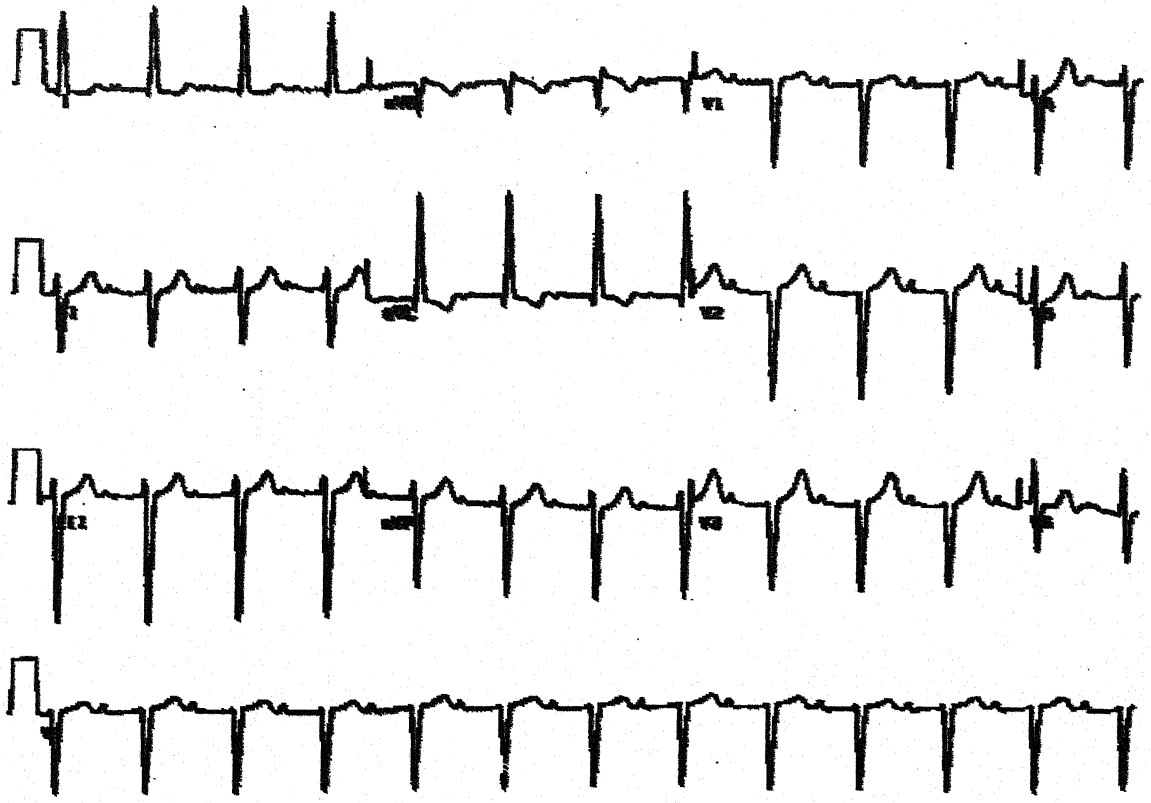


Fig. 6.1 : 12 Lead ECG signal of an 84 year old lady with hypertension

There are a number of abnormalities here.

- Left anterior hemiblock
  - QRS axis more left than - 30 degrees
  - initial R wave in the inferior leads (II, III and a VF)
  - absence of any other cause of left axis deviation
- Left ventricular hypertrophy

- In the presence of left anterior hemiblock the diagnostic criteria of LVH are changed Rosenbaum suggested that an S wave in lead III deeper than 15 mm as predictive of LVH.

➢ long PR interval (also called first degree heart block)
- PR interval longer than 0.2 seconds

➢ left atrial hypertrophy
- M shaped P wave in lead II
- P wave duration > 0.11 seconds
- terminal negative component to the P wave in lead VI

2. A LADY WITH ROMANO-WARD SYNDROME

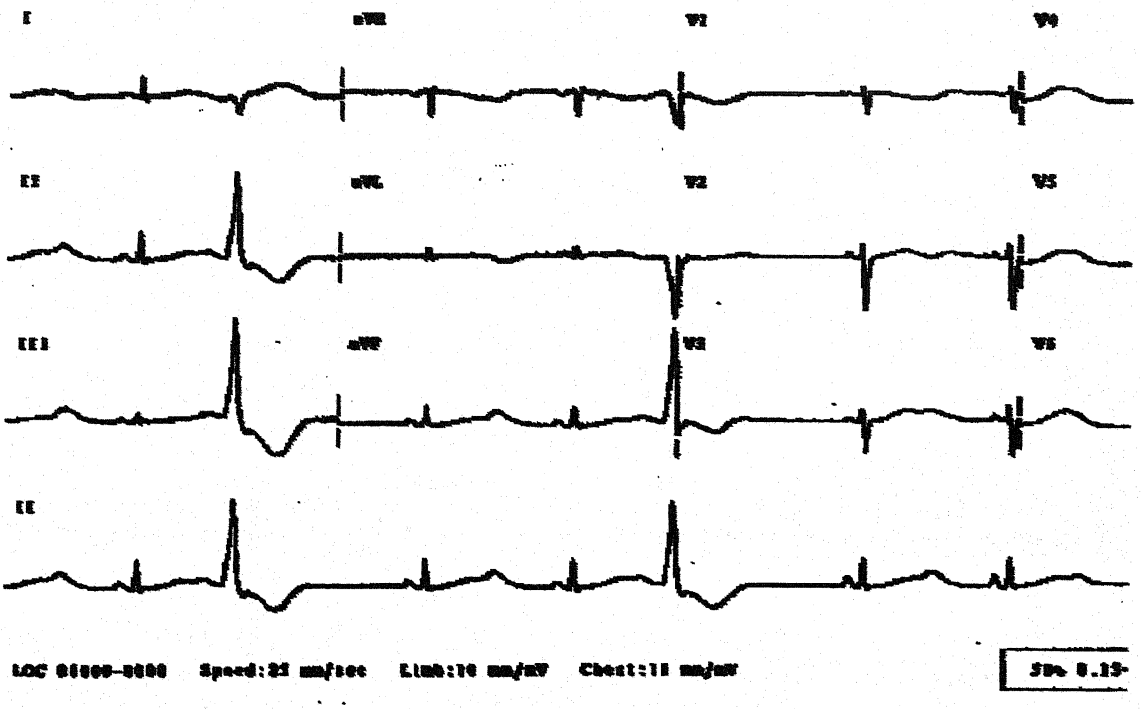


Fig. 6.2 : 12 Lead ECG signal of a lady with Romano-Ward Syndrome.

Long QT interval

- The QT interval normally varies with heart rate - becoming shorter at faster rates. It is usually corrected using the cycle length (R-R interval) as shown opposite.
- Normal QTc=0.42 seconds

- Romano-Ward syndrome is an autosomal dominantly inherited form of long QT interval and there is a risk of recurrent ventricular tachycardia, particularly Torsade de Pointes.

Ventricular premature beats (VPBs)

- 2 ventricular premature beats are also shown in this ECG
- They are
  - broad
  - Occur earlier than normal
  - and are followed by a full compensatory pause (the distance between the normal beats before and after the VPB is equal to twice the normal cycle length).

3. A NORMAL ADULT 12-LEAD ECG

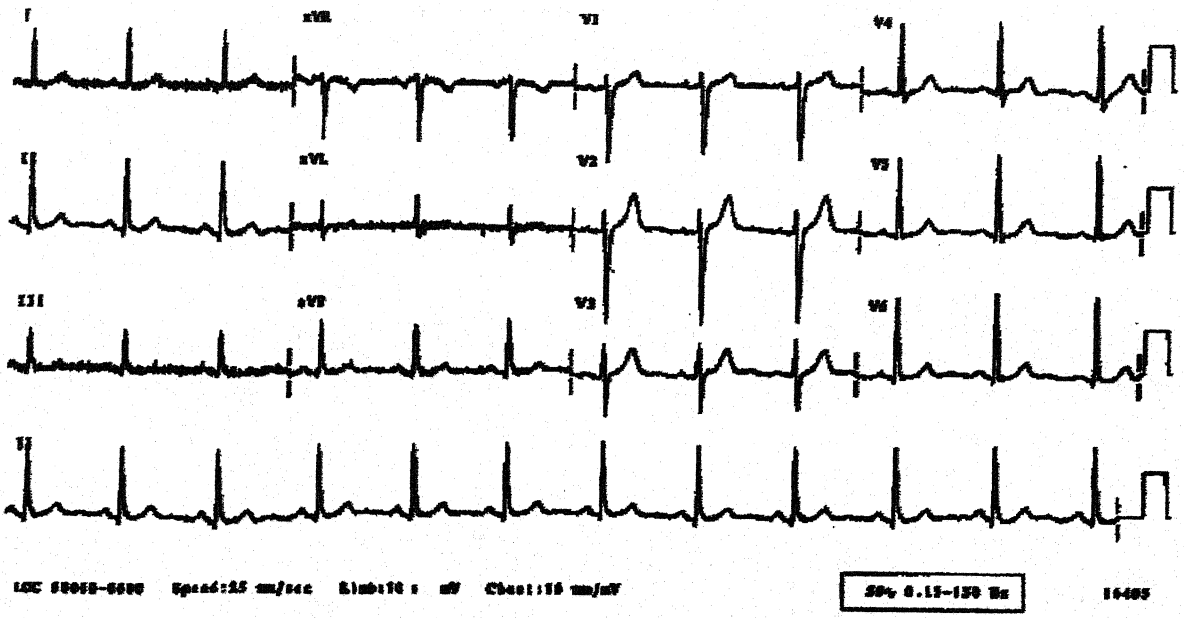


Fig. 6.3 : 12 Lead ECG signal of a normal adult

The diagnosis of the normal electrocardiogram is made by excluding any recognised abnormality. It's description is therefore quite lengthy.

- Normal sinus rhythm
- each P wave is followed by a QRS

- P waves normal for the subject
- R wave rate 60-100 bpm with <10% variation
  - Rate <60=sinus bradycardia
  - Rate >100 = sinus tachycardia
  - variation >10% = sinus arrhythmia
  -

4. A 47 YEAR OLD MAN WITH A LONG HISTORY OF PALPITATIONS AND, LATELY, BLACKOUTS.

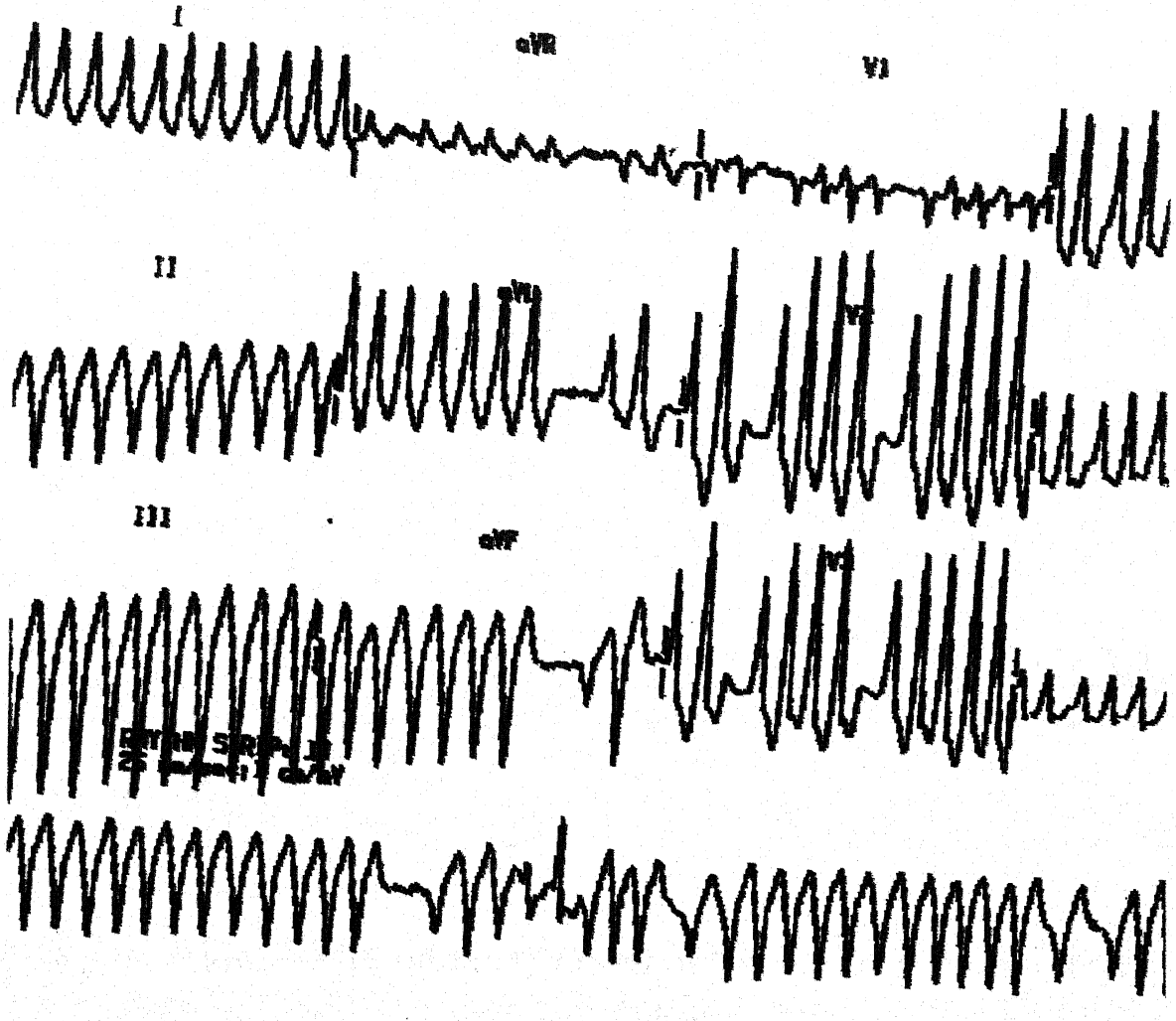


Fig. 6.4 : 12 Lead ECG signal of a 47 year old man with a long history of palpitations and lately, blackouts.

Wolf-Parkinson-White syndrome with atrial fibrillation

- irregularly irregular, wide complex tachycardia

- impulses from the atria are conducted to the ventricles via either
  - both the AV node and accessory pathway producing a broad function complex
  - or just the AV ode producing a narrow complex (without a delta wave)
  - or just the accessory pathway producing a very broad 'pure' delta wave
  - people who develop this rhythm and have very short R-R- intervals are at higher risk of VF.

5. A 45 YEAR OLD LADY WITH PALPITATIONS AND HISTORY OF CHRONIC RENAL FAILURE

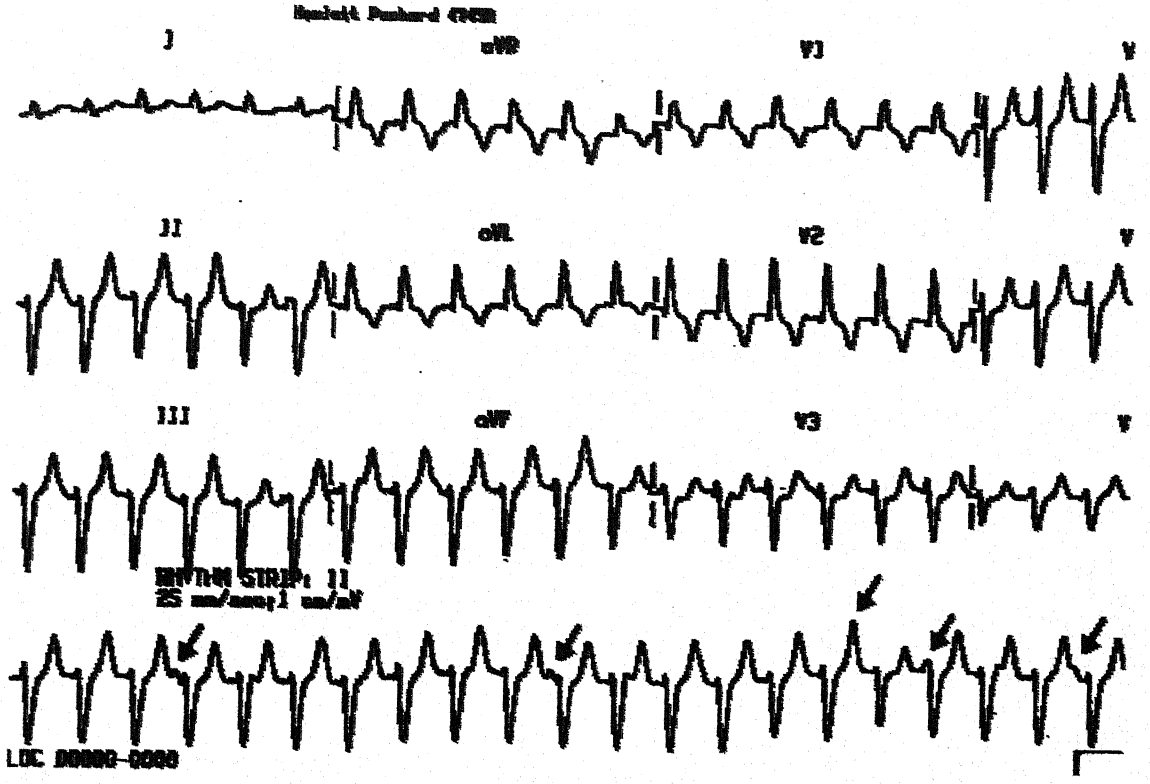


Fig. 6.5 : 12 Lead ECG signal of a 45 year old lady with palpitations and history of chronic renal failure

Ventricular tachycardia

A wide QRS tachycardia is VT until proven otherwise (1). Features suggesting VT include :-

- evidence of AV dissociation

- independent P waves (shown by arrows here)
- capture or fusion beats
- beat to beat variability of the QRS morphology
- very wide complexes (>140ms)
- the same morphology in tachycardia as in ventricular ectopics
- history of ischaemic heart disease
- absence of any rS, RS or Rs complexes in the chest leads
- concordance (chest leads all positive or negative)

6. A 72 YEAR OLD MAN WITH A PERMANENT PACEMAKER.

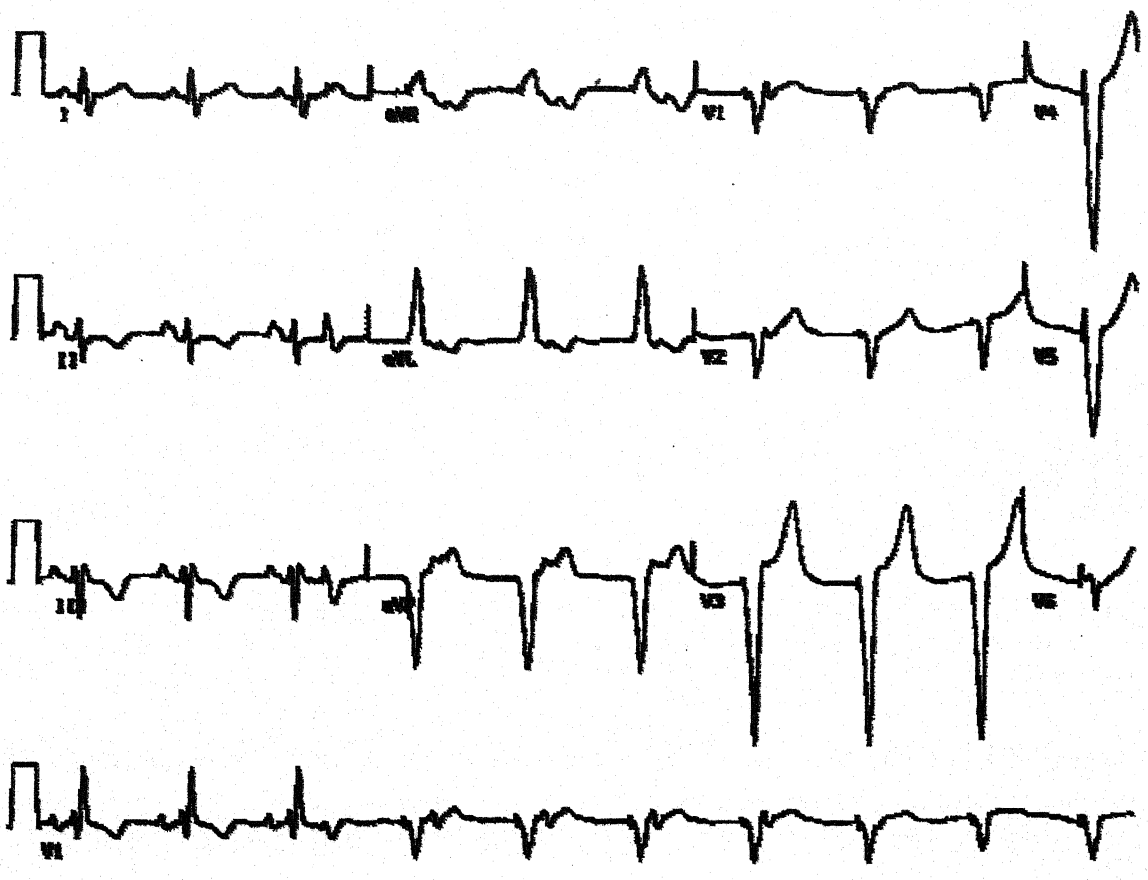


Fig. 6.6 : 12 Lead ECG signal of a 72 year old man with a permanent pacemaker

Ventricular pacemaker

- pacing spikes (best seen here in V4-V6) will be seen-they may be subtle
- the paced QRS complexes are abnormally wide

In this example the pacemaker starts when there is a long R-R interval following a blocked atrial premature beat (arrowed in figure). Sinus rhythm takes over again later in the rhythm strip.

7. A 60 YEAR OLD MAN WITH 2 HOURS OF "CRUSHING" CHEST PAIN SUDDENLY COLLAPSES

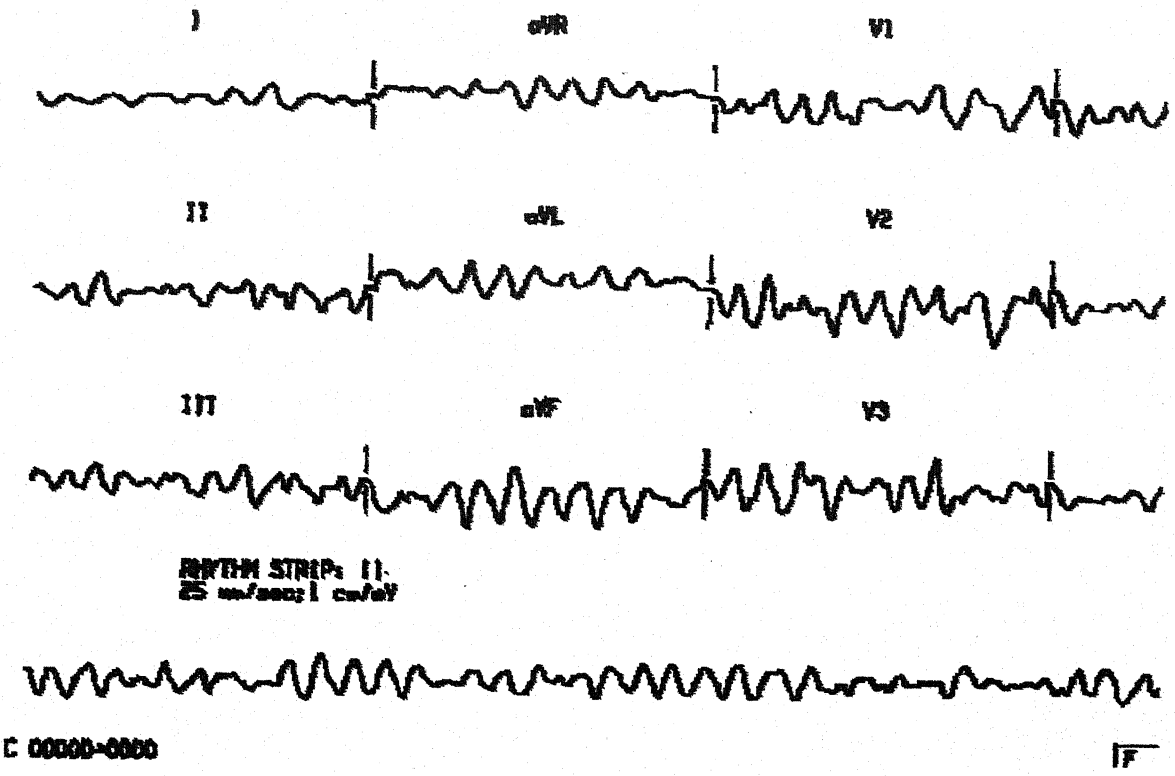


Fig. 6.7 : 12 Lead ECG signal of A 60 year old man with 2 hours of "crushing" chest pain suddenly collapses.

Ventricular fibrillation

- bizarre, irregular, random waveform
- no clearly identifiable QRS complexes or P waves
- wandering baseline

8. A 34 YEAR OLD LADY WITH ASTHMA

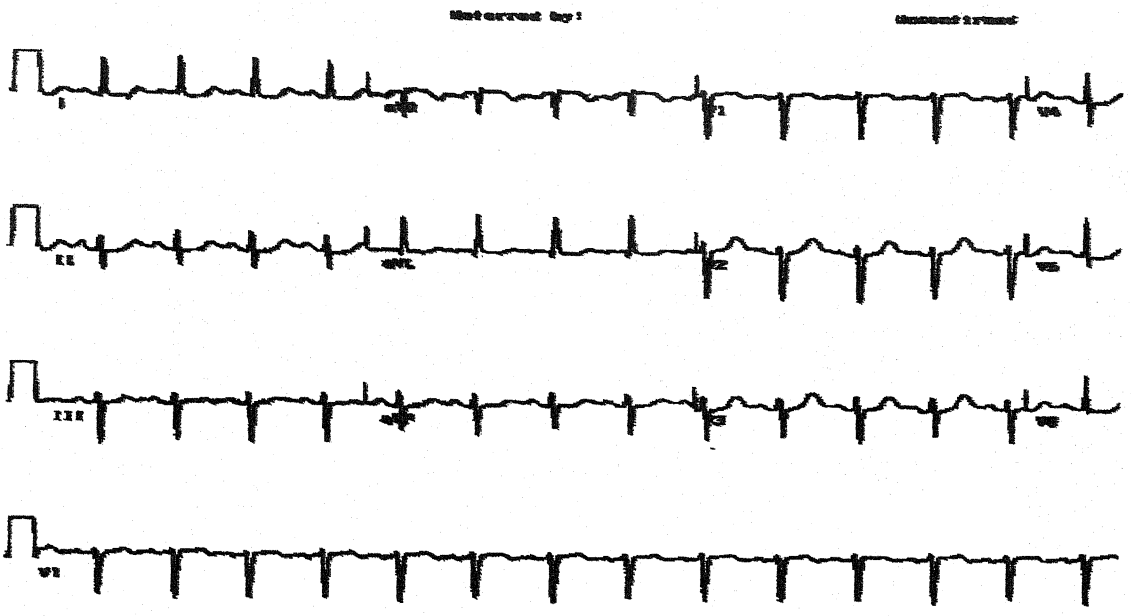


Fig. 6.8 : 12 Lead ECG signal of a 34 year old lady with asthma

Sinus tachycardia

- P wave rate greater than 100 bpm

9. A 55 YEAR OLD MAN WITH 4 HOURS OF "CRUSHING" CHEST PAIN

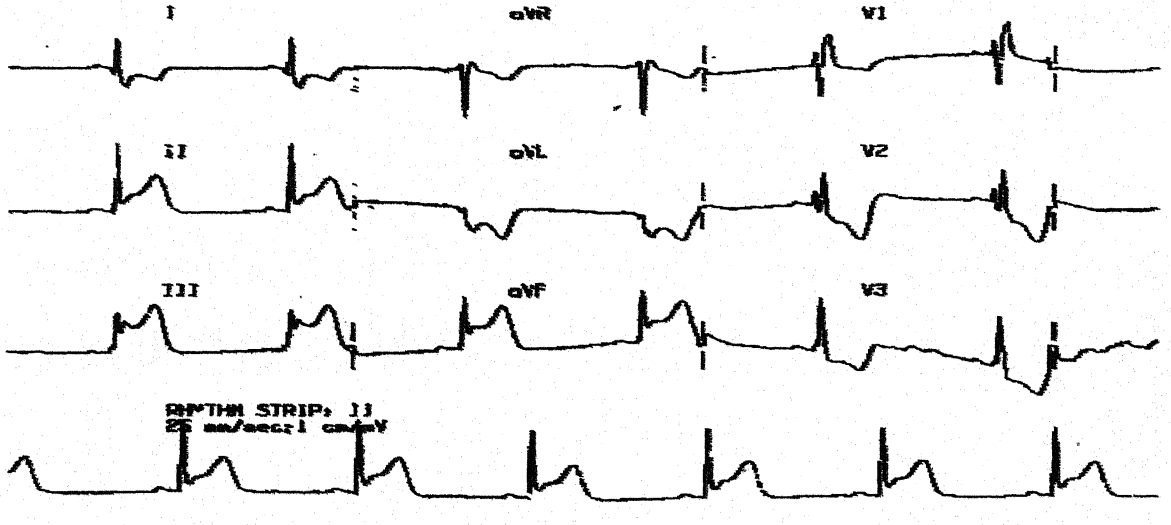


Fig. 6.9 : 12 Lead ECG signal of a 55 year old man with 4 hours of

"crushing" chest pain

Sinus bradycardia

- P wave rate of less than 60 bpm
- the rate in this example is about 45 bpm

See also Sinus Tachycardia

Acute inferior MI and Right Bundle Branch Block are also present.

10. A 59 YEAR OLD LADY WITH CHRONIC BRONCHITIS

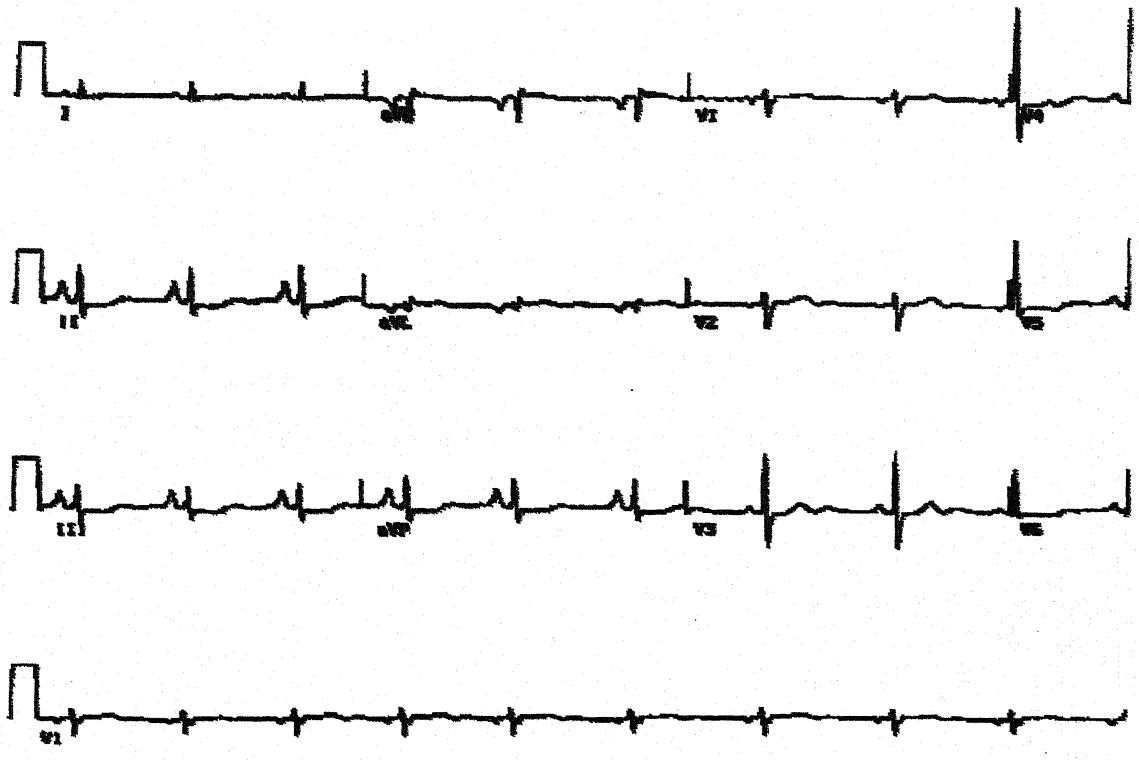


Fig. 6.10 : 12 Lead ECG signal of a 59 year old lady with chronic bronchitis

Right atrial hypertrophy

- A P wave in lead II taller than 2.5 mm (2.5 small squares).
- The P wave is usually pointed.

11. A 76 YEAR OLD MAN WITH BREATHLESSNESS

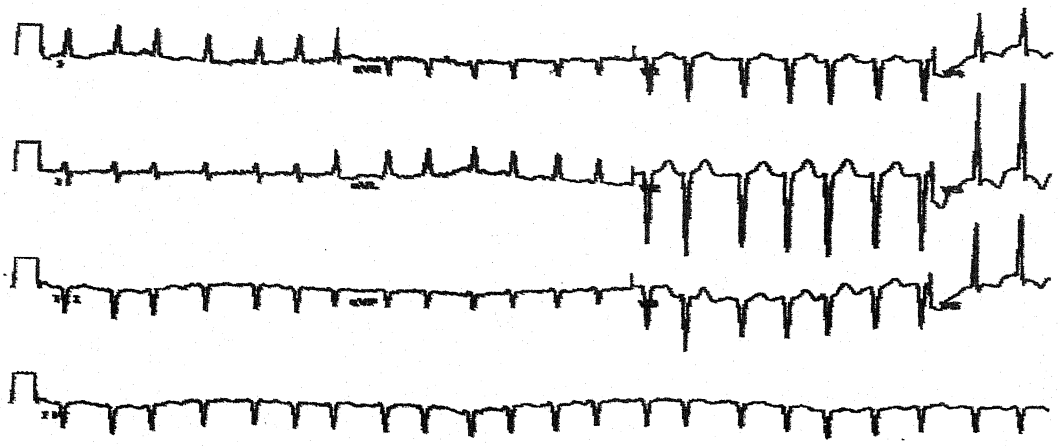


Fig. 6.11 : 12 Lead ECG signal of a 76 year old man with breathlessness.

Atrial fibrillation with rapid ventricular response

- Irregularly irregular ventricular rhythm
- Sometimes on first look the rhythm may appear regular but on closer inspection it is clearly irregular.

12. A 58 YEAR OLD MAN ON HAEMODIALYSIS PRESENTS WITH PROFOUND WEAKNESS AFTER A WEEKEND FISHING TRIP.

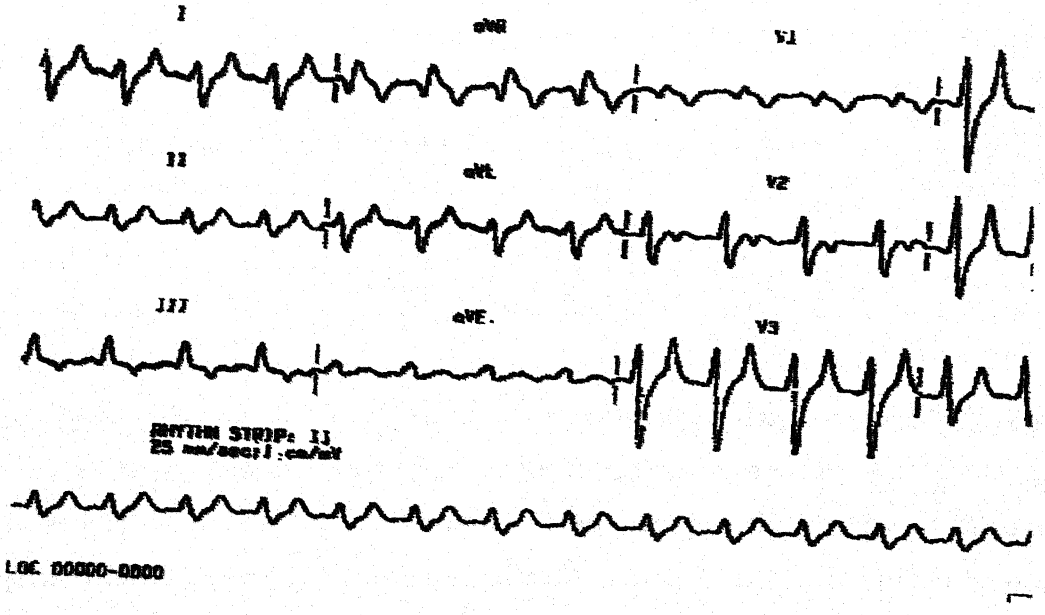


Fig. 6.12 : 12 Lead ECG signal of a 58 year old man on haemodialysis presents with profound weakness after a weekend fishing trip.

This man's serum potassium was 9.6 mmol/L

Hyperkalaemia

The following changes may be seen in hyperkalaemia

- small of absent P waves
- atrial fibrillation
- wide QRS
- shortened or absent ST segment
- wide, tall and tented T waves
- ventricular fibrillation

6.4 CONCLUSION

Diagnostic ECG interpretation requires high-quality signals (that is, noise-free signals with proper gain and band-width) recorded from several channels. Therefore the algorithms take into consideration the redundancy available in many leads. For example, the QRS complex may be isoelectric in one lead, but may be quite large in an orthogonal lead. Artifact is often not present in all the leads at the same time. This is because the artifact often arises from electrode failure or movement at some specific electrode site. Thus, artifact can be rejected by selectively processing the leads. The ECG interpretation algorithms, to a large extent, rely first on accurate QRS detection. Some arrhythmias such as bradycardia can be classified simply from beat-to-beat intervals. The QRS detector also helps localize the exact incidence in time of the Q, R, S, P, and T waves for later morphologic analysis. Ectopic beats, such as paced or fusion beats, may be classified on the basis of beat morphology. This also makes the task of detecting some arrhythmias, such as atrial fibrillation (characterized by high rate of atrial depolarization) or atrioventricular dissociation (discontinuous conduction between atria and ventricles), quite difficult. P-wave detection is crucial in identification of atrial disorders. Analysis of ST segment and the T wave is particularly useful in the detection of myocardial ischemia and infarction. Classification of more complex arrhythmias depends on a combination of rhythm and morphology analysis.

Thus the simulation of normal Human ECG and Arrhythmia is provided :

1. A ready database of Normal ECG and seventeen arrhythmias to analyze these data further to develop new techniques of analysis of Arrhythmias.

2. An easily operated window based simulation for teaching purpose and research purpose.

3. Noise free high quality signals at RA, LA, LL, V1, V2, V3, V4, V5, V6 for normal ECG and seventeen arrhythmias as Bradycardia, Tachycardia, Asystole, Fusion beat, Missed beat, Atrial fibrillation, Ventricular fibrillation, Bigeminy, Multifocal Ventricular Extrasystoles, R on T wave, Ventricular Tachycardia, Heartblock, VPB1 and VPB2.

4. The portable unit of ECG/Arrhythmia simulator battery operated so as to carry at any CCU to check the ECG/Arrhythmia monitor performance.

CHAPTER-VII

FURTHER SCOPE OF WORK

Diagnostic ECG interpretation requires high-quality signals (that is, noise-free signals with proper gain and band-width) recorded from several channels. Therefore the algorithms take into consideration the redundancy available in many leads. For example, the QRS complex may be isoelectric in one lead, but may be quite large in an orthogonal lead. Artifact is often not present in all the leads at the same time. This is because the artifact often arises from electrode failure or movement at some specific electrode site. Thus, artifact can be rejected by selectively processing the leads. The ECG interpretation algorithms, to a large extent, rely first on accurate QRS detection. Some arrhythmias such as bradycardia can be classified simply from beat-to-beat intervals. The QRS detector also helps localize the exact incidence in time of the Q, R, S, P, and T waves for later morphologic analysis. Ectopic beats, such as paced or fusion beats, may be classified on the basis of beat morphology. This also makes the task of detecting some arrhythmias, such as atrial fibrillation (characterized by high rate of atrial depolarization) or atrioventricular dissociation (discontinuous conduction between atria and ventricles), quite difficult. P-wave detection is crucial in identification of atrial disorders. Analysis of ST segment and the T wave is particularly useful in the detection of myocardial ischemia and infarction. Classification of more complex arrhythmias depends on a combination of rhythm and morphology analysis.

The software development process begins by devising either flowcharts or decision tables. Flowcharts are commonly employed in many software application where the computer goes through a series of logical actions. The program flow may depend on the result of specific tests. Such decision points are illustrated by diamonds in the flow-chart. The principal criteria for arrhythmia detection are R-R interval and heart rate. The flowchart distinguishes a few arrhythmia groups on the basis of these criteria. More relined discussion would require additional criteria and tests.

Diagnostic ECG interpretation deals with a large number of disorders, and requires more information than just the rate for decision making. For example, the pattern

of the ECG waveform itself must be analyzed in more detail. For example, analyzes parts of the QRS complex. On the basis of slope this table determines if the QRS complex has an RS or QR configuration.. Once again a decision table lists all the criteria associated with a specific group of disorders. When all the criteria are specified, a specific disorder such as acute inferior infarction is recognized. It becomes clear that a large number of such tables are required to diagnose important disorders that a detailed diagnostic ECG program should be able to interpret.

ECG/Arrhythmia simulation provides a wide database to develop Computer-aided analysis for the following:

1. ECG filtering and artifact rejection
2. QRS detection and rate calculations
3. Identification of P and T waves and ST segments
4. Ectopic beat identification (PVC, paced, fusion. etc.)
5. Arrhythmia classification based on rate (bradycardia, tachycardia, etc.)
6. Classification of disorders based on morphology (hypertrophy, infarct. etc.)

The microcontroller based ECG /Arrhythmia simulator consisted of two main sections viz. hardware and software sections. Both the sections were implemented successfully and checked on the storage oscilloscope as well as on the ECG monitor. The optimum utilisation of the microcontroller memory is done. So small size, portability, cost effectiveness of the simulator added further advantages to the instrument.

Though at present 17 arrhythmias and one normal ECG are considered, the number of the arrhythmias can be extended by using the extra two keys provided in the present hardware. Beside this, the hardware can be interfaced with the Computer to obtain the signals and can be further used for analysis purpose which would not be possible as always the data of arrhythmia patient is rarely obtained.

This can be further developed using VLSI Technology. Its size is further reduced as well as its usage at different ECG / Arrhythmia monitors is enhanced.

The database provided by this simulation is further used to analyse with MATLAB Tools for analysis purposes using Neurocomputing algorithms and fuzzy tools.

A Windows based software ECGSIM can further be developed to simulate atrial conditions and His and purkinje fibres simulation.

Thus ECG / Arrhythmia simulation has a wide scope in medical topic, electrocardiology which is attracting the early interest of engineers.

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9. Shahanaz Ayub, paper 'Myoelectric arm using Piezoceramics', National Seminar on Advances in Electroceramics(NSAE-2006), DRDO centre for piezoceramics and devices, Pune on 5th & 6th May, 2006

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APPENDIX I

SPECIFICATIONS :

Output Amplitudes	High level output	low level output
	1 V +/- 1%	1 mV +/- 3%
All waveforms	1.5 V +/- 3%	1.5 mV +/- 3%
	2 V +/- 8%	2 mV +/- 8%

Rate accuracy (all waveforms) : 3%

Waveform selection (one push-button per waveform) :

Normal	Atrial Fibrillation	Bradycardia
VPB 1	Ventricular Fibrillation	Tachycardia
VPB 2	Ventricular Tachycardia	Heartblock
Asystole	R on T Wave	Bigeminy
Paced	Run of multifocal VPBs	Fusion Beat
Couplet	Run of four VBBs	Missed Beat

Outputs :

*. Available through convenient terminals

*. 10 convenient terminals for checking 12 Lead ECG machine.

*. High level output via 10 pin FRC connector

*. Display(LED) indication for each selection of waveform

Power ON indication : Green LED glows

Battery : 6 V/ 1.2 Ah , Lead acid rechargeable.

Weight (with battery) : 500 gms

Dimensions : 18 cm x 16 cm x 5 cm

CONNECTOR DETAILS :

CN1 : Flatcable connector = 20 pins

(to interconnect the two PCBS)

CN2: Flatcable connector = 10 pins

(all outputs 1 V to display on CRO)

CN3: Flatcable connector = 10 pins

(to connect 1 mV signals to front panel terminals)

Connector Pin Details :

NC	---------	1	20	---------	NC
P 2.7	---------	2	19	---------	P 1.0
P 2.6	---------	3	18	---------	P 1.1
P 2.5	---------	4	17	---------	P 1.2
P 2.4	---------	5	16	---------	P 1.3
P 2.3	---------	6	15	---------	P 1.4
P 2.2	---------	7	14	---------	P 1.5
P 2.1	---------	8	13	---------	P 1.6
P 2.0	---------	9	12	---------	P 1.7
GND	---------	10	11	---------	VCC

CN2 : 1 V O/P

V2	-----	1	10	-----	L
V6	-----	2	9	-----	V5
V1	-----	3	8	-----	V4
F	-----	4	7	-----	V3
GND	-----	5	6	-----	R

CN3 : 1 mV O/P

V3	-----	1	10	-----	V6
V2	-----	2	9	-----	V1
V5	-----	3	8	-----	V4
R	-----	4	7	-----	L
GND	-----	5	6	-----	F

BILL OF MATERIALS :

SR. NO.	DESIGNATION	DESCRIPTION	MAKE & TYPE	QUANTITY
1	R25,R26,R9,R10 R11,R12,R13,14 R15,R16,R17	RESISTOR 1K 0.4 W , 5%	PHILIPS SFR 25	10
2	R1,R2,R3	RESISTOR 10K 0.4 W , 5%	PHILIPS SFR 25	3
3	R22,R23,R24	RESISTOR 5K6 0.4 W, 5%	PHILIPS SFR 25	3
4	R20	RESISTOR 1M 0.4 W, 5%	PHILIPS SFR 25	1
5	R21	RESISTOR 100K 0.4 W, 5%	PHILIPS SFR 25	1
6	R18,R19	RESISTOR 4K7 0.4 W, 5%	PHILIPS SFR 25	2
7	R30,R35,R40 R45,R50,R55 R60,R65,R70	RESISTOR 22K 0.4 W, 5%	PHILIPS SFR 25	9
8	R31,R36,R41 R46,R51,R56 R61,R66,R71	RESISTOR 33K 0.4 W, 5%	PHILIPS SFR 25	9
9	R27,R32,R37 R42,R47,R52 R57,R62,R67	RESISTOR 68E 0.4 W, 5%	PHILIPS SFR 25	9
10	R28,R29,R33,34 R38,R39,R43,44 R48,R49,R53,54 R58,R59,R63 R64,R68,R69	RESISTOR 3K3 0.4 W, 5%	PHILIPS SFR 25	18
11	R72,R74	RESISTOR 1K5 0.4 W, 5%	PHILIPS SFR 25	2
12	R75 R73	RESISTOR 3K3 680E ,2W	PHILIPS SFR 25	1 1
13	RB1 RB2	RESISTOR 10K NETWORK 1K	WEBEL SE8 472G	1 1
14	C4,C7,C10,C13 C16,C19,C22,25 C28,C6,C5,C9,8	CAPACITOR MULTILAYER CERAMIC	GUJRAT MULCO EL - LTD HCO8-103-M01	27

BILL OF MATERIALS :

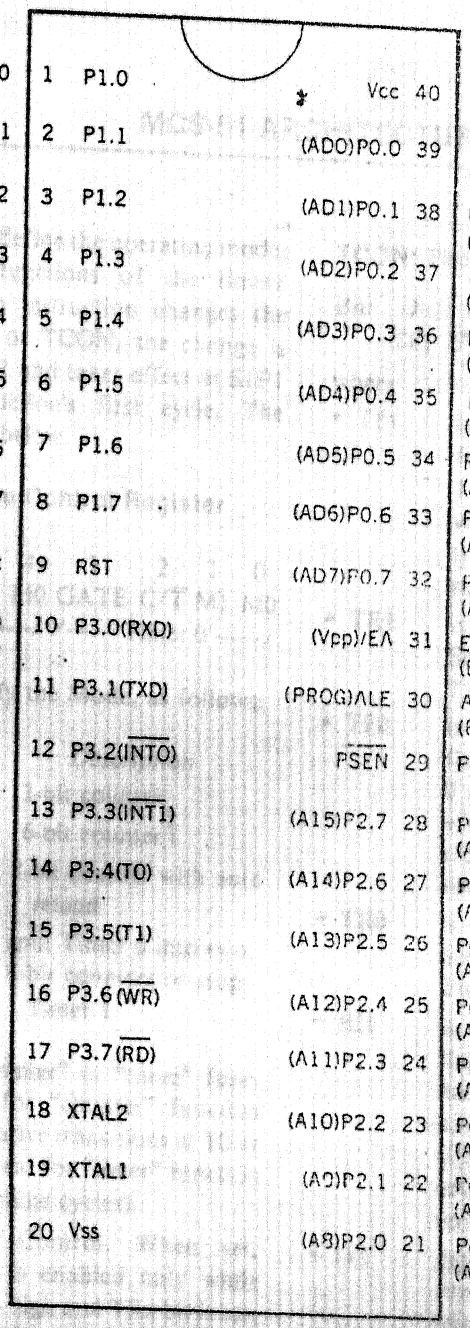
SR. NO.	DESIGNATION	DESCRIPTION	MAKE & TYPE	QUANTITY
14	C12,C11,C15, C14,C16,C17 C21,C20,C24 C23,C27,C26 C30,C29	0.1 MICROFARAD		
15	C1,C2	CAPACITOR CERAMIC 5pF,1 KV	GUJRAT MULCO EL - LTD	3
16	C3	CAPACITOR ELECTROLYTIC 10 microF/16V	KELTRON DB	1
17	C31	100microF/16V	KELTRON DB	1
18	C33,C34	CAPACITOR ELECTROLYTIC 2200uF/16V	KELTRON DB	2
19	C35	CAPACITOR BOXTYPE 0.22uF		1
20	Q5	TRANSISTOR PAD	JAIN	1
21	Q5	TRANSISTOR NPN	BC 107	1
22	X1	CRYSTAL 11.05924 MHz	KDS	1
23	P1,P2	POTENTIOMETER 100K	BORNS,3386 PACKAGE	2
24	P13	5K		1
25	P3	POTENTIOMETER 2K	BORNS, 3386 PACKAGE	1
26	L1 TO L18	LED RED 3mm	KWALITY KL 33 RD	19
27	D1,D2,D3	DIODES 75V	CDIL BA 159	3
28	IC9,10,11,12, IC13,14,15,16 IC17,IC8	IC SOCKETS (8 PINS)	O/E/N 8604,308-14-30	10
29	IC3	IC SOCKET (20 PINS)	O/E/N 8604,320-14-30	1

BILL OF MATERIALS :

SR. NO.	DESIGNATION	DESCRIPTION	MAKE & TYPE	QUANTITY
30	IC2	IC SOCKET (24 PINS)	O/E/N 8604,624-14-30	1
31	IC1,IC6, IC7,IC5	IC SOCKETS (16 PINS)	O/E/N 8604,316-14-30	4
32	IC4	IC SOCKET (40 PINS)	O/E/N 8604,640-14-30	1
33	IC9,10,11,12 IC13,14,15,16 IC17,IC8	IC, DUAL OP AMP	NATIONAL SEMICONDUCTOR LF 353	10
34	IC3	BIDIRECTIONAL BUFFER	HARRIS 74LS245	1
35	IC2	4 TO 16 DECODER	HARRIS 74LS154	1
36	IC1	3 TO 8 DECODER	HARRIS 74HCT138	2
37	IC6,IC7	IC, ANALOG DEMULTIPLEXER	MOTOROLA CD 4051	2
38	IC5	IC, D TO A CONVERTER	INTERSIL AD 7523	1
39	IC4	MICROCONTROLLER 8752 BH	INTEL 8752 BH	1
40	CN1	CONNECTOR 20 PINS M/F	O/E/N FRC	2
41	CN2,CN3	CONNECTOR 10 PINS M/F	O/E/N FRC	4
42	T1	TRANSFORMER	BIFILAR WOUND 18 TURNS PRIM SEC-2 WINDINGS	1
43	S1	SWITCH	SINGLE POLE 2 WAY (GILLARD)	1
44	S2	SWITCH	SINGLE POLE 3 WAY (GILLARD)	1

APPENDIX - II

8051 DIP Pin Assignments



Note: Alternate functions are shown below the port name (in parentheses). Pin numbers and pin names are shown inside the DIP package.

TCON, are used to define the operating modes and control the functions of the timer/counters. When an instruction changes the content of TMOD or TCON, the change is latched into the SFR and takes effect at S1P1 of the next instruction's first cycle. The registers are shown below.

TMOD: Timer Mode Control Register

Bit: 7 6 5 4 3 2 1 0
GATE C/$\overline{T}$ M1 M0 GATE C/$\overline{T}$ M1 M0
/----- Timer 1 -----/ /------ Timer 0 ------/

where M1, M0 specify the Mode, as follows:

M1	*M0*	*Mode*	*Description*
0	0	0	13-bit counter
0	1	1	16-bit counter
1	0	2	8-bit counter with auto reload
1	1	3	split Timer 0 into two 8-bit counters or stop Timer 1

- C/T selects "counter" or "timer" function. Set for "counter" function (count negative transitions at T0 or T1 pin). Clear for "timer" function (count machine cycles).
- GATE is Gating Control. When set, Timer "x" is enabled only while $\overline{INTx}$ pin is high and TRx bit is set. When cleared, Timer "x" is enabled whenever TRx bit is set.

Note: All bits of TMOD are cleared by reset.

TCON: Timer Control Register

Bit: 7 6 5 4 3 2 1 0
TF1 TR1 TF0 TR0 IE1 IT1 IE0 IT0

where

- TF1 is the Timer 1 overflow interrupt flag. Set by hardware when Timer 1 overflows. Cleared by hardware when the processor transfers control to the interrupt service routine.
- TR1 is the Timer 1 run control bit. Set/cleared by software to turn Timer 1 on/off.
- TF0 is the Timer 0 overflow interrupt flag. Set by hardware when Timer 0 overflows. Cleared by hardware when the processor transfers control to the interrupt service routine.
- TR0 is the Timer 0 run control bit. Set/cleared by software to turn Timer 0 on/off.
- IE1 is the external interrupt 1 edge flag. If IT1 = 1, this bit is set by hardware when $\overline{INT1}$ is detected to have made a 1-to-0 transition. Cleared by hardware when the processor transfers control to the interrupt service routine.
- IT1 determines whether external interrupt 1 is edge-triggered or level-triggered. If IT1 = 1, external interrupt 1 is edge-triggered. If IT1 = 0, external interrupt is triggered by a detected low at $\overline{INT1}$ rather than a 1 in IE1.
- IE0 is the external interrupt 0 edge flag. If IT0 = 1, this bit is set by hardware when $\overline{INT0}$ is detected to

INTERSIL

AD7523
8 Bit Monolithic Multiplying A/D Converters

FEATURES

- 8, 9 and 10 bit linearity
- Low gain and linearity Tempcos
- Full temperature range operation
- Full input static protection
- DTL/TTL/CMOS compatible
- +5 to +15 volts supply range
- Fast settling time: 100 nS
- Four quadrant multiplication
- 883B Processed versions available

GENERAL DESCRIPTION

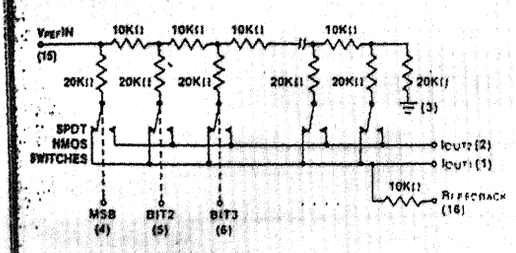
The Intersil AD7523 is a monolithic, low cost, high performance, 10 bit accurate, multiplying digital-to-analog converter (DAC), in a 16-pin DIP.

Intersil's thin-film resistors on CMOS circuitry provide 8-bit resolution (8, 9 and 10-bit accuracy), with DTL/TTL/CMOS compatible operation.

Intersil AD7523's accurate four quadrant multiplication, full military temperature range operation, full input protection from damage due to static discharge by clamps to V+ and GND and very low power dissipation make it a very versatile converter.

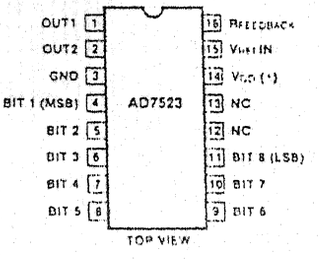
Low noise audio gain control, motor speed control, digitally controlled gain and attenuators are a few of the wide number of applications of the 7523.

FUNCTIONAL DIAGRAM



---- (Switches shown for Digital Inputs "High")

PIN CONFIGURATION

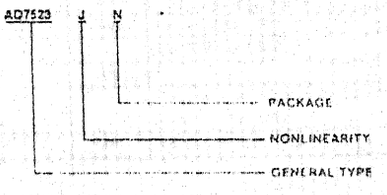


TOP VIEW

ORDERING INFORMATION

Nonlinearity	Temperature Range		
	0°C to +70°C	-25°C to +85°C	-55°C to +125°C
0.2% (8 Bit)	AD7523JN	AD7523AD	AD7523SD
0.1% (9 Bit)	AD7523KN	AD7523BD	AD7523TD
0.05% (10 Bit)	AD7523LN	AD7523CD	AD7523UD

PACKAGE IDENTIFICATION



SUFFIX D: CERDIP PACKAGE
SUFFIX N: PLASTIC PACKAGE

4

ABSOLUTE MAXIMUM RATINGS

(T_A = 25°C unless otherwise noted)

VDD (to GND) ..... +17V
VREF (to GND) ..... ±25V
Digital Input Voltage Range ..... -0.3 to VDD
Output Voltage Compliance ..... -0.3 to VDD
Power Dissipation (package)
Plastic
up to +70°C 670mW
derates above +70°C by ..... 8.3mW/°C
Ceramic
up to 75°C 4
derates above 75°C by 6m
Operating Temperatures
JN, KN, LN Versions ..... 0°C to +
AD, BD, CD Versions ..... -25°C to +
SD, TD, UD Versions ..... -55°C to +1
Storage Temperature ..... -65°C to +1
Lead Temperature (soldering, 10 seconds) ..... +3

CAUTION: 1. The digital control inputs are zener protected, however, permanent damage may occur on unconnected units under high e electrostatic fields. Keep unused units in conductive foam at all times.
2. Do not apply voltages higher than VDD and lower than GND to any terminal except VREF.

SPECIFICATIONS (VDD = +15V, VREF = +10V unless otherwise specified)

PARAMETER		TA +25°C	TA MIN-MAX	UNITS	LIMIT	TEST CONDITIONS
DC ACCURACY (Note 1)						
Resolution		8	8	Bits	Min	
Nonlinearity (Note 2)	(±1/2 LSB)	±0.2	±0.2	% of FSR	Max	
	(±1/4 LSB)	±0.1	±0.1	% of FSR	Max	-10V ≤ VREF ≤ +10V
	(±1/8 LSB)	±0.05	±0.05	% of FSR	Max	V_{OUT1} = V_{OUT2} = 0V
Monotonicity		Guaranteed				
Gain Error (Note 2)		±1.5	±1.8	% of FSR	Max	Digital inputs high
Nonlinearity Tempco (Note 2 and 3)		2		PPM of FSR/°C	Max	10V VREF ≤ +10V
Gain Error Tempco (Note 2 and 3)		10		PPM of FSR/°C	Max	
Output Leakage Current (either output)		±50	±200	nA	Max	V_{OUT1} = V_{OUT2} = 0
AC ACCURACY (Note 3)						
Power Supply Rejection (Note 2)		0.02	0.03	% of FSR/%	Max	V_{DD} = 14.0 to 15.0V
Output Current Settling Time		150	200	nS	Max	To 0.2% of FSR, R_L = 100Ω
Feedthrough Error		±1/2	±1	LSB	Max	VREF = 20V pp, 200KHz sine wave, digital inputs low
REFERENCE INPUT		5K		Ω	Min	
Input Resistance (Pin 15)		20K		Ω	Max	All digital inputs high (I_{OUT1} at grou
Temperature Coefficient (Note 3)		-500		ppm/°C	Max	
ANALOG OUTPUT (Note 3)						Both outputs
Voltage Compliance (Note 4)		-100mV to V_{DD}				See maximum ratings
Output Capacitance	C_{OUT1}	100		pF	Max	All digital inputs high (VINH)
	C_{OUT2}	30		pF	Max	
	C_{OUT1}	30		pF	Max	All digital inputs low (VINL)
	C_{OUT2}	100		pF	Max	
DIGITAL INPUTS						
Low State Threshold (VINL)		0.8		V	Max	Guarantees DTL/TTL and CMOS (0.5
High State Threshold (VINH)		2.4		V	Min	max, 14.5 min) levels
Input Current (per input)		±1		μA	Max	V_{IN} = 0V or +15V
Input Coding		Binary/Offset Binary				See Tables 1 & 2
Input Capacitance (Note 3)		4		pF	Max	
POWER REQUIREMENTS						Accuracy is tested and guaranteed at
Power Supply Voltage Range		+5 to +16		V		V_{DD} = +15V, only.
IDD		100		μA	Max	All digital inputs low or high.

NOTES: 1. Full scale range (FSR) is 10V for unipolar and ±10V for bipolar modes.
2. Using internal feedback resistor, $R_{FEEDBACK}$.
3. Guaranteed by design; not subject to test.
4. Accuracy not guaranteed unless outputs at ground potential.

Specifications subject change without not

ABSOLUTE MAXIMUM RATINGS

Supply Voltage
- LT1070/71HV (See Note 1) 60V
- LT1070/71 (See Note 1) 40V

Switch Output Voltage
- LT1070/71HV 75V
- LT1070/71 ..... 65V

Feedback Pin Voltage (Transient, 1ms) ..... ± 15V

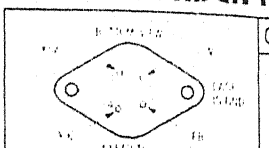
Operating Junction Temperature Range
- LT1070/71HVM, LT1070/71M ..... −55°C to +150°C
- LT1070/71HVC, LT1070/71C (Oper.) ..... 0°C to +100°C
- LT1070/71HVC, LT1070/71C (Sh. Ckt.) ... 0°C to +125°C

Storage Temperature Range ..... −65°C to +150°C

Lead Temperature (Soldering, 10sec) ..... 300°C

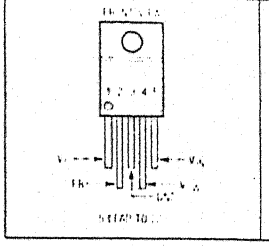
Note 1: Minimum switch "on" time for the LT1070/LT1071 in current limit is ≈ 1.0μsec. This limits the maximum input voltage during short circuit conditions, *in the buck and inverting modes only*, to ≈ 35V. Normal (unshorted) conditions are not affected. Mask changes are being implemented which will reduce minimum "on" time to ≤ 1μsec, increasing maximum short circuit input voltage above 40V. If the present LT1070/LT1071 (contact factory for package date code) is being operated in the buck or inverting mode at high input voltages and short circuit conditions are expected, a resistor must be placed in series with the inductor, as follows:

PACKAGE/ORDER INFORMATION



ORDER PART NUMBER

LT1070/LT1071HVMK
LT1070/LT1071MK
LT1070/LT1071HVCK
LT1070/LT1071CK



LT1070/LT1071HVCT
LT1070/LT1071CT

The value of the resistor is given by:

$$R = \frac{t * f * V_{IN} - Vf}{I_{(LIMIT)}} - R_L$$

t = Minimum "on" time of LT1070/LT1071 in current limit, ≈ 1μs

f = Operating frequency (40kHz)

Vf = Forward voltage of external catch diode at $I_{(LIMIT)}$

$I_{(LIMIT)}$ = Current limit of LT1070 (≈ 8A), LT1071 (≈ 4A)

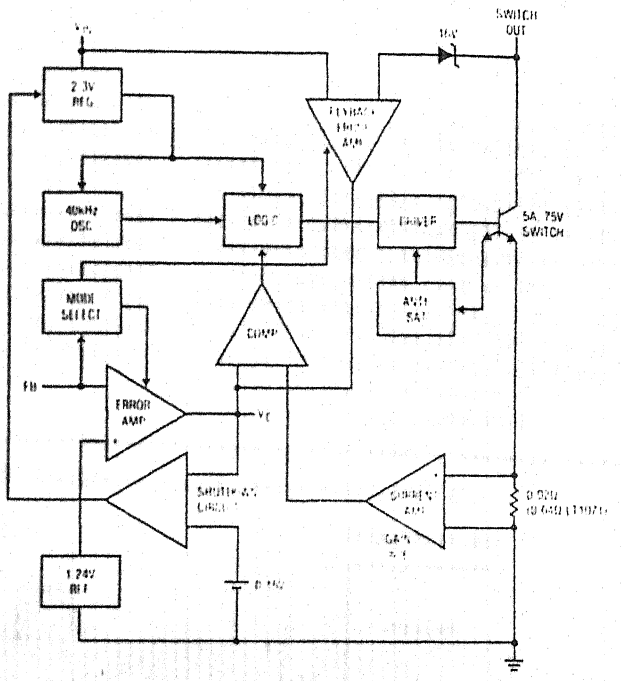
R_L = Internal series resistance of inductor

ELECTRICAL CHARACTERISTICS

Unless otherwise specified, V_{IN} = 15V, V_C = 0.5V, V_{FB} = V_{REF}, output pin open.

SYMBOL	PARAMETER	CONDITIONS		MIN	TYP	MAX	UNITS
V_{REF}	Reference Voltage	Measured at Feedback Pin		1.224	1.244	1.264	V
			●	1.214	1.244	1.274	
I_B	Feedback Input Current	$V_{FB} = V_{REF}$			350	750	nA
			●			1100	
gm	Error Amplifier Transconductance	$\Delta I_C = \pm 25\mu A$		3000	4400	6000	μmho
			●	2400		7000	
	Error Amplifier Source or Sink Current	V_C = 1.5V		150	200	350	μA
			●	120		400	μA
	Error Amplifier Clamp Voltage	Hi Clamp, V_{FB} = 1V		1.6		2.3	V
		Lo Clamp, V_{FB} = 1.5V		0.25	0.38	0.52	V
	Reference Voltage Line Regulation	$3V \le V_{IN} \le V_{MAX}$	●			0.03	%/V
A_V	Error Amplifier Voltage Gain	$0.7V \le V_C \le 1.4V$		500	800	2000	V/V
	Minimum Input Voltage		●		2.6	3.0	V
I_Q	Supply Current	$3V \le V_{IN} \le V_{MAX}$, V_C = 0.6V			6	9	mA
	Control Pin Threshold	Duty Cycle = 0		0.8	0.9	1.08	V
			●	0.6		1.25	
	Normal/Flyback Threshold on Feedback Pin			0.4	0.45	0.54	V
V_{FB}	Flyback Reference Voltage	I_{FB} = 50μA		15	16.3	17.6	V
			●	14		18	

BLOCK DIAGRAM



LT1070/LT1071 OPERATION

The LT1070/LT1071 is a current mode switcher. This means that switch duty cycle is directly controlled by switch current rather than by output voltage. Referring to the block diagram, the switch is turned "on" at the start of each oscillator cycle. It is turned "off" when switch current reaches a predetermined level. Control of output voltage is obtained by using the output of a voltage sensing error amplifier to set current trip level. This technique has several advantages. First, it has immediate response to input voltage variations, unlike ordinary switchers which have notoriously poor line transient response. Second, it reduces the 90° phase shift at midfrequencies in the energy storage inductor. This greatly simplifies closed loop frequency compensation under widely varying input voltage or output load conditions. Finally, it allows simple pulse-by-pulse current limiting to provide maximum switch protection under output overload or short conditions. A low-dropout internal regulator provides a 2.3V supply for all internal circuitry on the LT1070/LT1071. This low-dropout design allows input voltage to vary from 3V to 60V with virtually no change in device performance. A 40kHz oscillator is the basic clock for all internal timing. It turns "on" the output switch via the logic and driver circuitry. Special adaptive anti-sat circuitry detects onset of saturation in the power switch and adjusts driver current instantaneously to limit switch saturation. This minimizes driver dissipation and provides very rapid turn-off of the switch.

A 1.2V bandgap reference biases the positive input of the error amplifier. The negative input is brought out for output voltage sensing. This feedback pin has a second function: when pulled low with an external resistor, it programs the LT1070/LT1071 to disconnect the main error amplifier output

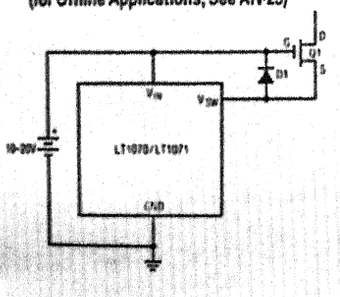
LT1070/LT1071 OPERATION

and connects the output of the flyback amplifier to the comparator input. The LT1070/LT1071 will then regulate the value of the flyback pulse with respect to the supply voltage. This flyback pulse is directly proportional to output voltage in the traditional transformer coupled flyback topology regulator. By regulating the amplitude of the flyback pulse, the output voltage can be regulated with no direct connection between input and output. The output is fully floating up to the breakdown voltage of the transformer windings. Multiple floating outputs are easily obtained with additional windings. A special delay network inside the LT1070/LT1071 ignores the leakage inductance spike at the leading edge of the flyback pulse to improve output regulation.

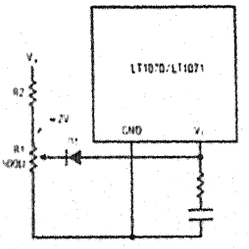
The error signal developed at the comparator input is brought out externally. This pin (V_C) has four different functions. It is used for frequency compensation, current limit adjustment, soft starting, and total regulator shutdown. During normal regulator operation this pin sits at a voltage between 0.9V (low output current) and 2.0V (high output current). The error amplifiers are current output (gm) types, so this voltage can be externally clamped for adjusting current limit. Likewise, a capacitor coupled external clamp will provide soft start. Switch duty cycle goes to zero if the V_C pin is pulled to ground through a diode, placing the LT1070/LT1071 in an idle mode. Pulling the V_C pin below 0.15V causes total regulator shutdown, with only 50μA supply current for shutdown circuitry biasing. See AN-19 for full application details.

TYPICAL APPLICATIONS (Note that maximum output currents are divided by 2 for LT1071.)

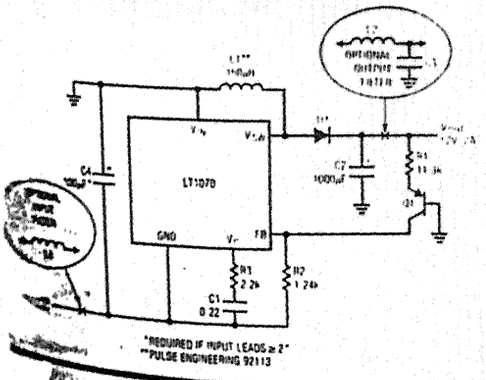
Driving High Voltage FET
(for Offline Applications, See AN-25)



External Current Limit



Negative to Positive Buck-Boost Converter



External Current Limit